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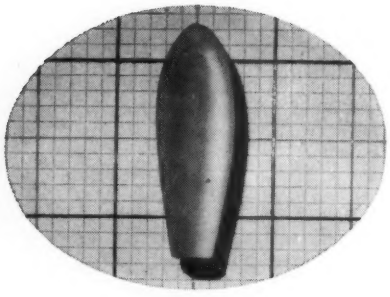

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The Manitoba Medical Review

Vol. 36 FEBRUARY, 1956 No. 2

Obstetrics & Gynecology

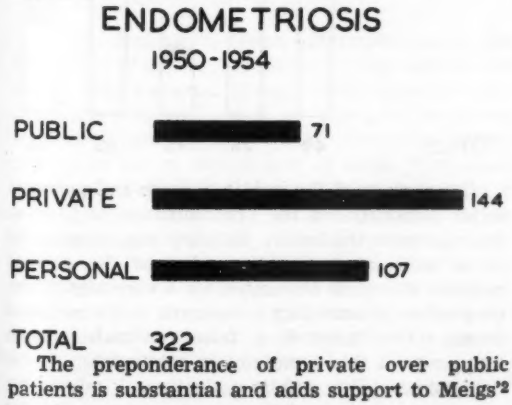
Endometriosis

D. E. Cannell, M.B., and F. H. Munkley, M.D.

Since Sampson[1] first drew attention to endometriosis as a clinical and pathological entity much has been written in regard to its histogenesis, clinical characteristics and management. The frequency of its occurrence — its etiology, treatment and histogenesis have occasioned much discussion and some controversy. Our purpose today is to consider some features of this condition as it has been observed on the public and private gynaecological services of the Toronto General Hospital in the past 5 years and in a 15 year review of those cases occurring in the private practice of one of the authors.

Material

This report comprises a total of 322 patients who were found to have endometriosis. All cases are included in which there was clinical, operative or histological evidence of endometriosis. It is realized that this may confuse, rather than clarify, our understanding of the condition. It is difficult, if not impossible, in such a study to be sure whether endometriosis or the associated conditions cause the patient's disability. We have divided our patients into 3 groups — public, private and personal. The bulk of them were confirmed at laparotomy and by histological examination. These 3 groups, as a whole, are considered to give a broader understanding of the significance of endometriosis; considered singly, they tend to overemphasize certain features of the condition and thus distort the picture. Table I.



The preponderance of private over public patients is substantial and adds support to Meigs'[2] view in regard to the more frequent occurrence of this condition in women of higher economic status. A more accurate estimate of this disproportion is suggested by the 2:1 ratio at the Toronto General Hospital rather than 3.6:1 ratio of the whole series.

Incidence

An accurate estimation of the incidence of endometriosis was possible for only three years of this study. Table II. This demonstrates a greater frequency in the personal as opposed to the public series. The higher ratio of public and personal cases found at laparotomy, and confirmed pathologically, may be due to closer liaison between the surgeon and pathologist in these groups as opposed to that in the private cases.

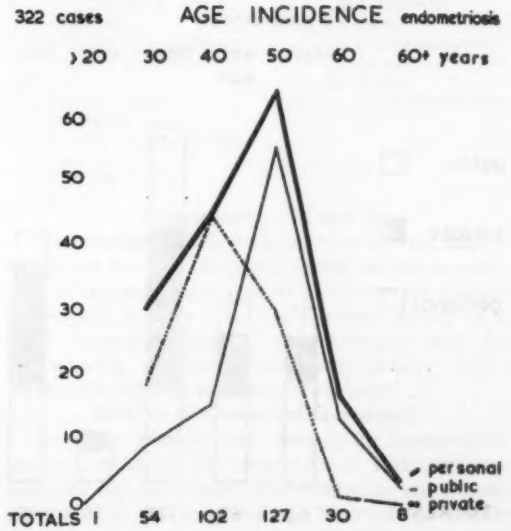
Table II

INCIDENCE

	Cases	Admiss'ns	Ratio to 1	Laparot.	Ratio 1 to
Public	56	3584	64	472	8
Private	101	3429	34	1138	11
Personal	21	507	24	70	3.3
Totals	178	7520	42	1680	9

Age — Table III

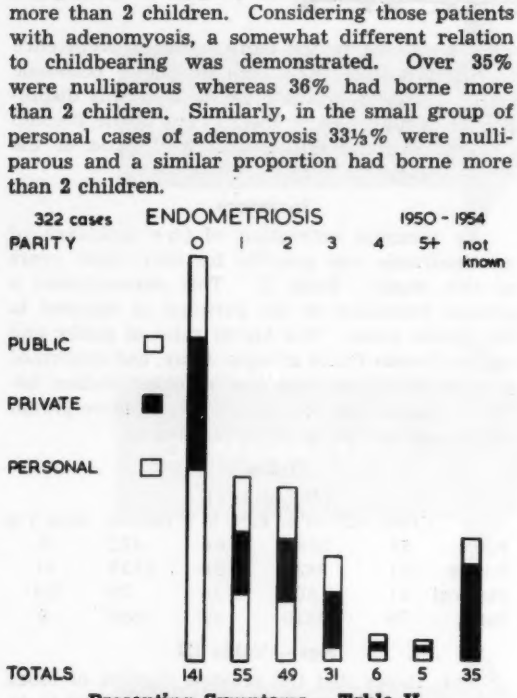
This shows that the greatest number of cases, 71%, developed in the two decades from 30 to 50, 1 occurred in the teens, 54 in the 20's and 38 in the 50's and 60's. It is suggested that in many more instances the condition is present but undiagnosed in the 20's.



From the Toronto General Hospital and Department of Obstetrics and Gynaecology, University of Toronto.
Presented at the Sectional Meeting of the American College of Surgeons, Winnipeg, April 25th, 1955.

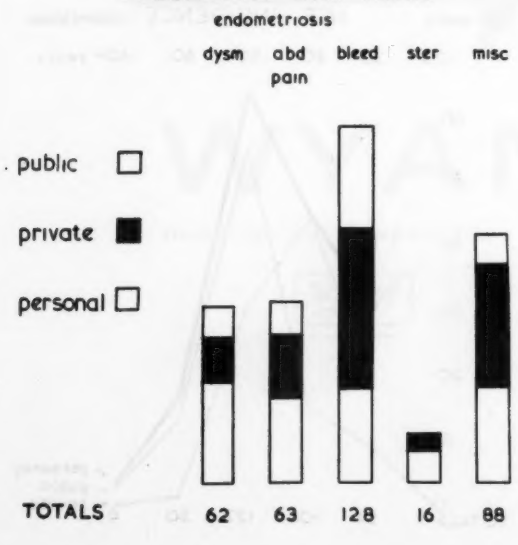
Parity — Table IV

In the whole group over 40% of the patients were nulliparous and 13% had had more than 2 children. In the personal series almost 60% were nulliparous and slightly more than 6% had borne more than 2 children. Considering those patients with adenomyosis, a somewhat different relation to childbearing was demonstrated. Over 35% were nulliparous whereas 36% had borne more than 2 children. Similarly, in the small group of personal cases of adenomyosis 33⅓% were nulliparous and a similar proportion had borne more than 2 children.



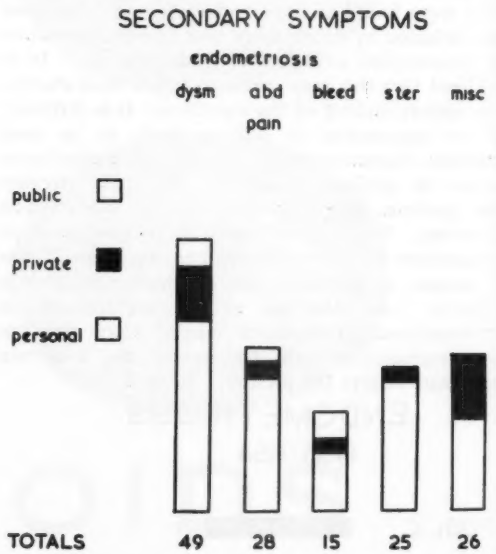
Presenting Symptoms — Table V

Gynaecological patients present themselves most frequently with complaints of vaginal bleeding or pain. Patients in this study complained of bleeding, miscellaneous symptoms, abdominal pain, dysmenorrhea and sterility in this order. The miscellaneous group presented a bizarre picture, none of which, with the possible exceptions of dyspareunia and rectal pain, bore any pathognomonic relation to endometriosis. If one considers abdominal pain and dysmenorrhea together, pain is as frequent as bleeding as a primary symptom.



Some contrast between presenting complaints in the public and private patients as opposed to those in the personal series are worthy of comment. Bleeding was more frequent in the former, whereas dysmenorrhea was most frequent and considered together with abdominal pain, occurred almost twice as frequently as bleeding in the personal group. Sterility in the public and private series represented less than 3% of the primary complaints whereas it constituted over 9% of the personal cases.

Secondary complaints in the whole group (Table VI) were predominantly those of dysmenorrhea and abdominal pain, whereas the occurrence of bleeding was insignificant.

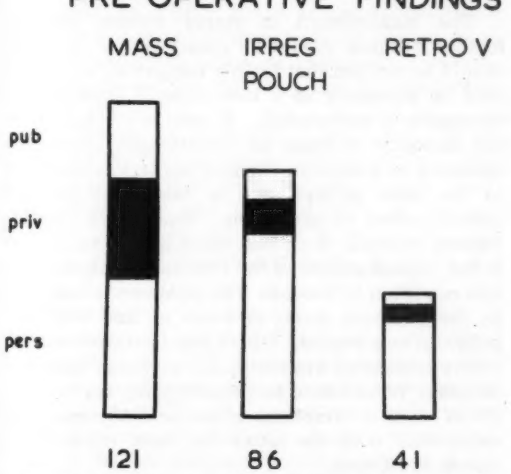


Comparison of the public, private and personal series demonstrated the predominance of pain as a symptom in the latter. Sterility was complained of in none of the public and 1 of the private patients though it accounted for a very significant proportion of secondary complaints in the personal series. The latter is a fairer estimate of the frequency of this complaint in relationship to endometriosis. Few public patients seek advice for sterility and many of the private cases would not be admitted to hospital unless surgical treatment were required.

Diagnosis Table VII

The preoperative findings are shown in this table. It is impossible to assess the frequency of correct preoperative diagnoses in the private series as all our information is based upon hospital histories and a disproportionate number of correct diagnoses were made without the confirmation of pelvic examination.

Here again, a significant difference between the public, private and personal cases is noted. Irregularity in the fornices or pouch of Douglas constitute 21% of the former and 43% of the personal groups. Irregular utero-sacral studding is one of the earliest findings in endometriosis and careful examination with this in mind usually reveals it. Associated fixed retroversion was present in only 6% of the private and public patients, whereas it was found in 23% of the personal group. This may be explained on the basis of the incompleteness of the sample of private and public cases.



It was possible to obtain information as to the frequency of correct preoperative diagnoses in the public and personal group. The diagnosis was made preoperatively in 16 instances on the public service, at laparotomy in 14, and was an incidental finding in 28; in comparison 18 cases were diagnosed preoperatively, 18 at laparotomy and 14 were incidental findings in the personal series. Histological confirmation of the diagnosis was positive in all but 5 of these 108 cases. Microscopic corroboration of the operative diagnosis will be obtained more frequently if the surgeon marks the suspected area by suture, or better still, cuts a block for the pathologist's attention.

Sites of Endometriosis — Table VIII

External endometriosis was the commonest finding at laparotomy. The adnexa were the most frequent site of disease. Adenomyosis was present

Table VIII

SITE OF LESION

	INTERNAL	EXTERNAL				
	Slight	Mod &	Adnexal	Misc.	Comb	Bowel
Public	8	20	36	4	2	1
Private	33	14	58	25	9	6
Personal	2	12	29	24	6	1
Totals	43	46	133	53	17	8

in 89 instances next in frequency to the adnexa. Other sites of external endometriosis such as the utero-sacral ligaments and pelvic peritoneum were noted on 53 occasions. Combined internal and external endometriosis was present in 17 patients. Bowel, umbilicus, abdominal scars and other sites were not frequent though the bowel lesions in most instances were serious and will be considered separately later.

Associated Lesions — Table IX

Associated pathological conditions were found in 133 instances. It should be stated that in many of these, the associated lesion was of more significance than the endometriosis. The bulk of these were fibroids and endometrial hyperplasia. In 5 instances endometriosis was an incidental finding in relation to uterine malignancy. In 3, it was present with ovarian carcinoma. More significantly, carcinomatous change had occurred in 3 patients within endometriosal lesions. These together with 2 other cases of similar nature collected elsewhere will be discussed in greater detail.

One of the congenital anomalies was that of incomplete atresia of the vagina which had resulted in haematometra and haematocolpos. To some degree this patient simulated the experimental work of TeLinde and Scott.[3] The operative and pathological findings were those of widespread endometriosis of the ovaries.

Table IX

ASSOCIATED PATHOLOGY

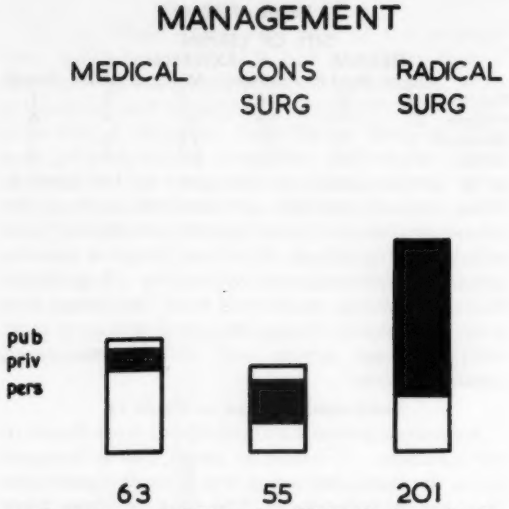
BENIGN	MALIGNANT
79 Fibroids	4 Ca. Body
18 Endo-M. Hyperplasias	1 Ca. in Adenomyosis
2 Cong. Anomalies	1 Ca. Cervix
10 P.I.D.	3 Ca. Ovary
4 Simple Cysts	2 Ca. in Endometriosis
3 Dermoids	
3 Serous Cystadenomas	
1 Pseudomucinous	
1 Fibroma Ovary	
1 Ectopic	

Management — Table X

The available data does not help a great deal in the problem of conservative versus radical surgery. 78% of the public and private patients were treated by radical surgery. In the personal series, 50% were treated medically; approximately 31% by radical, and 16% by conservative surgery — 2% were referred for treatment elsewhere.

Results of Conservative Surgery

In all, 55 patients were treated by conservative surgery, that is the childbearing function was theoretically preserved. The follow-up was incomplete in this group of cases. None of the public



ward cases were followed sufficiently long to determine the results satisfactorily. In the private and personal group, 21 were relieved of pain, 4 were unrelieved, 3 had insufficient follow-up. 8 were relieved of sterility and subsequently delivered 1 or more living children. The extent of disease and the operative procedures employed demonstrated no apparent relation to the success achieved. Again, in the personal series there was a higher proportion of success in both sterility and relief of pain than in the private series. This may be due to more prolonged and intensive follow-up and therapy.

Discussion

The series here reported provides a reasonable cross section of the problem, management and success to be anticipated in treatment of hospital and private patients suffering with endometriosis. The incidence of this condition is low as compared to some other reported series. This may be accounted for by the inclusion of public ward patients in the study and the failure to obtain histological confirmation of the diagnosis in all cases where it was present on the private service. The ratio of 1:3.3 found at laparotomy in the personal series is similar to that of Meigs and is probably a more accurate estimate of the incidence than that of the combined survey. There is no new information in regard to increased incidence of endometriosis in individuals of better economic status and decreased fertility. Our data suggest that these factors parallel Meigs' findings. Endometriosis is not characterized by more specific symptoms than those presented in other gynaecological conditions. The greater incidence of bleeding in the public and private cases may be due to a higher proportion of adenomyosis in this group. The occurrence of dysmenorrhea and abdominal pain, with much greater frequency in the personal series, is probably a more accurate indication of its frequency than that in the other groups. This may be explained by the fact that patients with minimal lesions, and often minor symptoms, sought attention, whereas only those with greater involvement were admitted to hospital in the other groups. Similarly, sterility as a primary symptom was more frequent in the personal series and is indicative of greater interest in this field of gynaecology. It does not imply that endometriosis per se is a frequent cause of infertility. Indeed, the etiological relationship of endometriosis to sterility is difficult to explain.

In regard to the physical findings, the importance of careful palpation of the pouch of Douglas, the utero-sacral ligaments and adnexa cannot be over emphasized. The earliest finding of minimal endometriosis is usually demonstrable by utero-sacral studding. It is impossible to differentiate preoperatively with any accuracy between adenomyosis and fibromyomata uteri. The former may be suspected if dysmenorrhea is a prominent symptom.

The management in young women desiring further children should be conservative. Patients should be warned that further surgery or treatment may be necessary at a later date if conservative treatment is undertaken. It was not required in the majority of cases as symptomatic relief was complete or adequate. Radical surgery is desirable in the older patient and in those who demand assured relief of symptoms. The larger number treated radically is an indication of this factor and is not representative of the overall management of this condition in Toronto. The treatment of patients in the personal series is more in line with our policy in this regard. Where non-operative conservative treatment was instituted we found that mild sedation, reassurance and observation were as useful in relief of symptoms as hormone therapy. Our experience with the latter has been too scant to assess its efficacy.

Two points of interest are worthy of extended consideration in this study.

(1) Endometriosis of Bowel

Eight cases were encountered in this study; 7 were significant lesions, 1 occurred during pregnancy. All were associated with complete or incomplete intestinal obstruction, 2 of small bowel, 5 of large bowel. The one occurring in pregnancy was relieved by lysis of adhesions and a plastic procedure on the small intestine. The remainder required resection and in only one instance was hysterectomy and bilateral salpingo-oophorectomy done. The lesions in the genital organs in these cases were presumably minimal, though it is difficult to determine this from the surgeon's operative reports. In no instance did the obstruction occur at the time of the menses. Except on two occasions there is little to indicate that endometriosis was

suspected prior to laparotomy. In a study of 19 cases of endometriosis of the bowel, which includes 7 of those reported here, Ryley[4] notes that 13 patients showed no demonstrable evidence of endometriosis elswhere. In 1, rectal bleeding occurred at the time of menstruation. In 5 of his cases barium enema revealed constricting lesions of the sigmoid and in 2 of these the radiological diagnosis was carcinoma. Sigmoidoscopic examination was useful in differential diagnosis in one instance. Counseller[5] has suggested that the diagnosis may be suspected where sub-acute or chronic obstruction occurs in females with a prolonged history of sterility and dysmenorrhea. In acute obstruction, unless there is a history of bowel haemorrhage at the menses or the obstruction occurs at that time, there is little to suggest endometriosis as a cause of obstruction. The management in the younger patients should be conservative in regard to genital lesions if further childbearing is desired; simple resection of the obstructive lesion is indicated. In the older patients both pelvic and bowel surgery is indicated. In neither group, where endometriosis of the bowel is of significant extent, should treatment by castration alone, be considered adequate. Post-menopausal scarring may lead to obstruction, if this is done.

The treatment in endometriosis of the bowel seems to have been adequate in these patients. Two required further treatment — 1 surgical, for recurrent obstruction and 1 radiological for vaginal bleeding. In 1 case carcinoma of the caecum was present, as well as endometriosis of ileum and sigmoid.

(2) Malignancy

Association of endometriosis and malignancy in this report is high. Eight cases of carcinoma elsewhere in the female genital organs were found, and in at least 2 of these the associated endometriosis was a significant finding. The occurrence of carcinoma in ectopic endometrium has been reported recently in increasing numbers. In this study, 2 others were found in adnexal lesions and 1 in adenomyosis. Two of these are being reported elsewhere by Kumar[6] — we wish to add a further 2 to the 16 of the 27 cases previously reported which he considers satisfy Sampson's criteria of malignancy occurring in endometriosis. Scott[7] suggests that we may have to revise our teaching that endometriosis is a completely benign lesion. In the light of reported experience this would seem sound. It is interesting to speculate upon the number of carcinomas of the ovary which have their origin in small ectopic endometrial lesions. Nothing here reported warrants the assumption that more radical surgical treatment is indicated in the overall management of endometriosis because of the possible occurrence of malignant change in this condition. It is our opinion that the indications for surgery in gynaecological disease are already too broad rather than too circumscribed. Morbidity and possibly mortality would be increased rather than decreased by such measures.

Conclusions

1. A series of 322 cases of endometriosis is here reported.

2. The observation of Meigs of the more frequent occurrence of this condition in the nulliparous patient of better economic status is supported by our experience.

3. Conservative treatment without hormonal administration is effective in many cases with minimal lesions.

4. Conservative surgery is justifiable in the younger age groups and is reasonably satisfactory in the relief of sterility.

5. Adequate resection of bowel lesions is indicated in all groups complicated by the occurrence of endometriosis in the gut. In the older patients castration as an additional procedure is suggested to eradicate the condition completely.

6. Malignant change does occur in endometriosis and 2 additional cases of such malignancy are here reported.

Acknowledgment

The authors wish to acknowledge the assistance of Dr. J. D. M. Gillies in the preparation of our tables, Drs. F. A. Jaffe and W. A. Anderson for the photomicrographs, Dr. L. T. Armstrong of the Toronto Western Hospital for permission to use his case of malignancy occurring in endometriosis and Drs. D. Kumar, T. G. Ryley and T. B. Robson for use of their material and criticism.

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Medicine

Iron Deficiency Anemia in a Case of Haemochromatosis Treated by Repeated Phlebotomy*

S. Vaisrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C) F.A.C.P.
W. Karlinsky, M.D., F.R.C.P. (C)

Davey, Foxell and Kemp[1] have recently reported a case of haemochromatosis in whom a total of 31 litres was removed over the course of two years. At the end of this period the patient developed a severe anaemia preceded by a low serum iron. This development suggested that, even though the estimated total body-iron stores (25-50 gm.) were unlikely to have been exhausted by the venesections, they were no longer available for the synthesis of haemoglobin, possibly due to the iron being deposited in large slowly mobilizable aggregates of haemosiderin. This case was apparently, the first of its kind recorded in the medical literature.

The following case is the second.

Case Report

Mr. D., age 50, bachelor, a carpenter by trade, was admitted on November 17, 1953, to Deer Lodge Hospital complaining of "sores" on his scalp and weakness of several months' duration. His past history revealed that in 1922 he had a penile chancre and positive serology for which he was treated for approximately one year with arsenicals, bismuth and mercury. Following this his serology became negative and has since remained negative. In 1945, while serving in England, he developed headache and blurred vision, and was recorded as having "macular degeneration." Blood and spinal fluid were normal. The headaches and blurred vision persisted for approximately 18 months and then disappeared.

In 1951, at which time he weighed 150 pounds, he developed thirst, polyuria and weight loss. In 1952 he noted a brownish discoloration of the skin of his legs, impotence, and impaired vision. In May, 1953, he developed "sores" on his scalp for which he consulted a physician, who diagnosed diabetes mellitus and instituted treatment.

His family history was as follows: His father died at the age of 50 of a "heart attack", his mother at the age of 40 following thyroid surgery. His paternal grandfather died at the age of 80, his maternal grandmother of hypertension at the age of 65. His maternal grandfather developed diabetes at the age of 60 and died at the age of 80. He recalls him having a "reddish" complexion.

The patient is the oldest of four children. The other three are females and are all alive and well. One step-sister is also well.

Physical examination on admission revealed a man 5 ft. 7 in. tall, weighing 115 pounds. His head hair was sparse and there was a healing superficial ulcer on his scalp. Heart and lungs were normal. Blood pressure was 110/80. The liver was firm and smooth and enlarged five fingers' breadth below the costal margin. The spleen was not palpable. The testicles were normal. The fundi showed early macular degeneration. Visual acuity was 20/25 in both eyes. There was an absence of axillary hair, and pubic and body hair were sparse. The extensor surfaces of the arms and legs showed a diffuse brownish pigmentation. The face also showed increased pigmentation, but here there was also a purplish-gray hue present.

Laboratory data were as follows: haemoglobin 90%, white blood count 12,000, sedimentation rate 41 mm. per hr.; urinalysis: sugar 1, albumen—faint trace; fasting blood sugar 185 mgm.%; total serum proteins 6.3 gm.% with 3.3 gm.% albumin and 3.0 gm.% globulin, serum bilirubin .77 mgm.%, blood urea nitrogen 14 mgm.%; intravenous bromsulfalein 21% retention after 30 minutes; Cephalin cholesterol flocculation 3+, thymol flocculation 3+, thymol turbidity 4.0 units, serum iron 280 micrograms %, 24 hour 17-ketosteroid output 8.2 mgm. Thorn test and Oleesky water test did not suggest adrenal hypofunction. Biopsy of the skin overlying one tibia was reported as follows: "Scattered throughout the deep corium are large fusiform cells containing brown pigment and spindle shaped cells with golden pigment. The latter cells reveal a distinct tendency to concentrate in the adnexal tissues and they stain intensely with Prussian blue. Melanin pigmentation appears normal."

Liver biopsy was reported as follows: "There is distortion of the lobular pattern by dense collagenous tissue bands in portal areas. There is a diffuse golden brown pigmentation of liver cells, and a concentration of this pigment in the Kupffer cells. The Prussian blue reaction on this pigment is strongly positive. Summary: haemochromatosis and cirrhosis."

On a 2,200 calorie diet and 25 units P.Z.I. his diabetes was reasonably well controlled with blood sugars ranging from 144 mgm.% to 170 mgm.%. During December, 1953, he had three separate phlebotomies of 400 cc. each. He was allowed out over the Christmas season with instruction to return before the year was out.

He was not seen until January 13, 1954, when he was brought in by ambulance. At this time he

*From the Faculty of Medicine, University of Manitoba, and the Department of Medicine, Deer Lodge Hospital, Winnipeg, Manitoba. Reprinted from Canadian Services Medical Journal, February, 1955.

was stuporous, dehydrated, afebrile, and had a right lobe pneumonia. His white blood count was 22,000 with 98% polymorphs. He was treated with penicillin—800,000 units daily and parenteral fluids. He remained critically ill for one week. At this time aureomycin, 1 gm. daily, and cortisone, 100 mgm. daily, were added and he began to improve within a few days. His weight in the meantime had dropped to 99 pounds. His fasting blood sugars ranged from 150-300 mgm.% on 25 units P.Z.I. and a 2,600 calorie diet.

A consistent program of repeated phlebotomy in order to deplete body-iron stores was begun on the 11th of May, 1954, and continued up to March, 1955, when a drop in the level of haemoglobin necessitated a prolonged interruption. At the commencement of this treatment, his weight was 105 pounds. His liver edge was five fingers' breadth below the costal margin. His haemoglobin was 79%, serum protein 6.5 gm.%, and serum iron 482 micrograms.%. His bone marrow was normal.

From May 13, 1954, to March 11, 1955, a total of 26 phlebotomies of approximately 600 cc. each were carried out. The dates of the venesections and their reflection on his haemoglobin, haematocrit, red blood count, serum proteins and fasting serum iron levels are recorded in Table I.

Phlebotomy 600 cc.	Date	Hb. in % 15.5gm. 100%	Haemato-crit	RBC (in millions)	Serum Proteins Total (gm. %)	Serum Iron in micrograms %
1.	13/5/54	79	32	3.9	6.5	----
2.	17/5/54	----	33	----	----	340
3.	21/5/54	----	32	----	----	----
4.	26/5/54	----	29	----	6.1	425
5.	31/5/54	----	32	----	5.2	315
6.	5/6/54	75	35	4.1	5.8	----
7.	11/6/54	80	33	3.6	6.0	----
8.	16/6/54	75	35	3.7	6.0	----
9.	22/6/54	83	40	4.3	6.1	305
10.	26/6/54	80	36	4.0	5.8	----
11.	30/6/54	75	36	4.0	----	----
12.	5/7/54	78	35	3.9	5.8	140
13.	12/7/54	78	37	4.1	6.3	105
14.	17/7/54	75	38	3.6	----	----
15.	23/7/54	80	40	----	6.1	170
16.	29/7/54	74	36	3.7	6.0	190
17.	4/8/54	72	35	3.5	6.1	60
18.	10/8/54	69	34	3.6	6.3	----
19.	20/8/54	75	35	3.7	6.1	----
20.	7/9/54	72	34	4.0	5.6	45
21.	18/9/54	72	32	3.8	6.5	----
22.	2/11/54	74	----	5.0	6.5	----
23.	12/11/54	71	----	----	----	----
24.	26/11/54	69	----	----	6.3	----
	9/12/54	56	----	----	----	----
25.	17/12/54	65	----	----	5.8	25
	7/1/55	64	----	4.4	----	----
	21/ /55	62	----	----	----	----
	25/2/55	65	----	----	----	----
26.	11/3/55	75	----	3.5	----	60
	28/3/55	51	----	3.9	----	----
	29/4/55	65	----	----	----	----
	20/5/55	64	----	----	----	----
	9/6/55	66	----	3.7	----	----
	22/7/55	76	----	----	----	----
	5/8/55	83	----	----	----	----

These 26 phlebotomies represent 12.6 litres of blood, more than twice his circulating blood volume at any one time. If one assumes that his average haemoglobin during this period was 11 gm.% then he was depleted of 4.62 g. of iron. This is more than the total iron content of normal man, but less than 20% of the estimated iron in a patient with haemochromatosis.

During this period of time it was necessary to increase his insulin dose from P.Z.I. 40 units before breakfast to his present dose of 60 units N.P.H. insulin before breakfast and 50 units N.P.H. insulin before supper. His fasting blood glucose values fluctuated from 130 to 250 mgm.% and his 24 urinary glucose output from a trace over 100 gm. It was noted, however, that there has been less fluctuation in these values during the latter part of this regime. His weight increased over a period of 14 months from 105 to 146 pounds. This very gratifying increase in weight was associated with increased strength and a sense of well being.

His brom-sulfalein retention decreased from 21% at onset of venesections to 12½% after 26 phlebotomies. His cephalin cholesterol flocculation remained strongly positive. There was no change in the size of the liver. The pigmentation of face, arms and legs has decreased markedly.

The serum proteins did not show any significant change, and it was never necessary to postpone phlebotomy because of a low value. It was, however, necessary to interrupt it for six months because of the fall in the haemoglobin to the low level of 51% in March, 1955.

Concomitant with the decrease in the haemoglobin was that of serum iron, which fell from 425 mgm.% (May 26, 1954) to 60 mgm.% March 11, 1955). His total iron binding capacity on the latter date was 428 mgm.%, iron unsaturation 368 mgm.% and iron absorption after four hours 225 mgm.%. The low serum iron and its high absorption and unsaturation indicated that the anaemia was that of iron deficiency.

Venesection was interrupted for six months. It was resumed in August, 1955, when the haemoglobin rose to 85%.

Discussion

The case of Mr. D. is, in its clinical aspects, a typical one of primary idiopathic haemochromatosis. Mr. D. presents the classical quadrad of diabetes mellitus, cirrhosis of the liver, pigmentation of the skin, and hypogonadism, as well as the characteristic asthenia. He lives up to the expectations of the laboratory with his high serum, iron content, positive skin, liver and bone marrow biopsies for iron, along with the characteristic laboratory findings of diabetes and cirrhosis. He even conforms, up to a point, to the expected clinical improvement following repeated phlebotomy.

Where Mr. D. departs from the usual (if one may be permitted the use of the term in a disorder as unusual as haemochromatosis) case is in the development of anaemia with low serum iron after venesections. This is an unexpected sequel. The usual response to the removal of blood in these cases is an increase in the haematopoietic activity of the bone marrow, with iron for the synthesis of haemoglobin drawn from body-iron stores. This,

indeed, is the rationale of the treatment, which aims at the depletion of the large amount of iron that has accumulated in the bodies of these patients throughout the years by dint of its excessive absorption through the gut due to an inborn disorder of the haemostatic mechanism ("mucosal block") that governs the absorption of iron.

Treatment of primary idiopathic haemochromatosis by repeated phlebotomy introduced independently by W. D. Davis[2] and his co-workers in 1947, and C. A. Finch and his group in 1948[3] has resulted in subjective and objective improvement in most cases thus treated. There was observed, along with the marked amelioration of symptoms, a clearing of the complexion, a recession in the size of the liver, and a diminution in the amount of iron deposited in the latter.[4] In no case, except the one reported by D. A. Davey[1] and that of Mr. D., did anaemia with low serum iron develop as a result of the treatment. Since the amount of iron removed from the patient treated by Davey (15 gm.) and from Mr. D. (5 gm.) was nowhere near the estimated total body iron stores, one may infer that in these cases the iron deposits are poorly mobilizable, being possibly fixed as large aggregates of haemosiderin. One may also prophesy that, unless a potent chelating agent can be found to mobilize the fixed iron (versene has been tried without too much success)[5], no further dramatic improvement will result from repeated phlebotomy in this type of case. The best that can be hoped for is a retardation of the progress of disease by phlebotomy repeated at infrequent intervals to forestall the development of a severe anaemia.

Summary

A case of primary idiopathic haemochromatosis, in whom repeated phlebotomy resulted in an anaemia with a low serum iron, was described. Therapeutic implications of the concept of fixed iron stores were discussed in brief.

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Case Report

Refractory Anemia With Spontaneous Recovery and Transfusion Hemochromatosis

W. J. Bennett, M.D.

Refractory anemia affecting the erythron only, as distinct from aplastic anemia, which involves all the blood elements, is an extremely rare condition. It is said that, if a patient afflicted with this disorder lives long enough, he will recover. The following is a case in point: the patient recovered after being maintained on transfusions for a period of five years.

In addition to the rare primary condition, this case presents the interesting complication of secondary hemochromatosis.

C. R., a 57 year old unmarried Canadian, an army veteran with a right hemiplegia and aphasia, resulting from "meningitis" contracted while on Service in 1916, was admitted to Deer Lodge Hospital on June 28, 1948 by ambulance from a lakeside resort 60 miles north of Winnipeg. Because of the aphasia, the history of onset of the present illness at the age of 50 was difficult to elicit, but it was determined that he had been his usual self until one week prior to admission when he caught a "cold", lost his appetite, began to feel weak, became short of breath and in general felt very ill, the symptoms developing gradually over the seven day period. He had a past history of treatment in a Sanatorium for far advanced pulmonary tuberculosis from 1932 to 1934, but he had experienced no symptoms ascribable to tuberculosis since that time. Enquiry elsewhere brought to light that despite the severe disability imposed by the hemiplegia, he had managed to get around quite well. It was later observed that he was of cheerful mien, alert, intelligent, emotionally stable with a spirit of independence and a constant merry twinkle in his eyes. He played cribbage well, using the left hand to hold and play the cards. Since discharge from the Sanatorium he had lived with a married sister in Winnipeg and was on a holiday when his illness began.

Physical examination showed a pale, dyspneic man in obvious distress. There was a hemiplegia of the right side with marked flexion deformity of the upper limb, the fingers and wrist in flexion and the hand drawn up and resting on the chest just below the chin. The lower limb was spastic and in internal rotation. His word power was limited to "Yes" and "No" and a repetitive jargon with which he tried to answer questions. The sclerae and conjunctivae were nearly white. Temperature was normal, pulse 120 and regular, respirations 40 per minute, blood pressure 150/70. There were no other physical findings of note. The hemoglobin was 18%, the red cell count 600,000. Blood Group A, Rh positive. He was given 1,000 ccs. of blood and felt improved though the hemo-

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globin rose to only 25% and the red blood cells to one million. Chest film showed clear fields and a cardio-thoracic ratio of 17/28 cms., the position of the right forearm and hand partly obscuring the right lung field. Scout film of the abdomen was normal; sigmoidoscopy to eight inches was negative, stools were negative for occult blood and no abnormality was noted in the barium enema. Gastric analysis showed 40 units of free hydrochloric acid, barium series and electrocardiogram were normal. Urinalysis was negative, the B.U.N. within normal limits, blood serology negative, and there was no apparent infectious process. On July 2nd hemoglobin rose to 30%, erythrocytes 1.06 million, leucocytes 5600 — the platelets were normal but no reticulocytes were seen. The mean corpuscular volume was 96 cu. microns, the mean corpuscular hemoglobin 44 cu. microns, and the mean corpuscular hemoglobin concentration was 44. Bone marrow obtained from the sternum was reported as showing a moderate shift to the left with no suggestion that the smear resembled that seen in pernicious anemia. A diagnosis of aplastic anemia was made, but as repeated blood examinations never revealed any abnormality in the leucocytes, it was considered that the aplasia involved the erythropoietic tissue only. Prothrombin time and platelets remained normal with no reticulocytes being noted in any examination. During the following year he was maintained relatively symptomless on blood transfusions averaging about 500 ccs. twice a week, was given ferrous sulphate grs. X t.i.d. and 15 units of liver, intramuscularly, daily. On this regime the hemoglobin usually remained between 40 - 56% and the red blood cells 1.5 to 3.0 million per cu. mm., and he was able to be up and around without distress. During this period the hemoglobin did on occasion drop to 18% but was promptly raised to 40% by blood transfusion, the only procedure which appeared to be beneficial. Repeat bone marrow study 11 months later showed no change from the initial reading and other tests including serum bilirubin estimation, liver function study, serum proteins and other procedures were equally uninformative in establishing a diagnosis other than aplastic anemia selective for erythrocytes. On June 8, 1949 he was discharged from hospital to be followed in the Out-Patient Department and to receive weekly blood transfusions. The patient's co-operation was excellent. In August 1949 he was re-admitted to hospital because of extreme weakness. He required help to get around. Hemoglobin was 23%, leucocytes 7,700, and blood pressure 150/70. Liver and spleen were not palpable. There was moderate pitting edema of the legs. Re-investigation failed to cast any further light on the etiology of the anemia. A trial with cobaltous chloride, 3 ccs. t.i.d. after meals was attempted, but had to be abandoned because of severe nausea and vomiting. He was discharged and put on Out-Patient follow-up and treatment as before.

In May 1950 he was evacuated from his home due to flood conditions and re-admitted to hospital. Apart from his complexion, which was now almost lemon-colored, no other changes were noted. By December 1950 after blood transfusions over the previous two and a half years, the blood picture showed no change, but there was a slate-colored pigmentation of the face and exposed skin as well as in the palmar skin creases. In January 1951 biopsy of the skin of the left wrist showed hemosiderosis of the deep layers of the skin and of the sweat glands. Liver function study revealed a Thymol Turbidity of 3; Thymol Flocculation 2, Cephalin Flocculation 3, B.S.P. 16% of the dye retained in one half hour, and liver biopsy showed infiltration of the liver with hemosiderin and a slight increase in fibrous tissue, particularly in the portal areas. The Prussian blue reaction was positive. The sedimentation rate was 162 mm/hour which was not much faster than on former occasions. To the previous diagnosis was added hemochromatosis due to multiple blood transfusions. In March 1951 the serum iron was 347 gamma per cent (Normal — 125 - 175 gamma %) the iron saturation less than 50% and the serum iron combining capacity 300-400 gamma per cent. There was no essential change in his condition throughout the balance of 1951, the blood picture fluctuating as before.

In February 1952 he was admitted to the hospital with broncho-pneumonia. He was very ill, his face ashen grey, his hemoglobin was 12%, erythrocytes 1.1 million per cu. mm., leucocytes 19,000, neutrophils 88, lymphocytes 11 and monocytes 1, sedimentation rate 181 mm/hour. The broncho-pneumonia responded to penicillin and the anemia to blood transfusion. On discharge February 18, 1952 the hemoglobin was 47 per cent. When he was examined on March 17, 1952, the hemoglobin was 54% and it was the first time since the onset of the anemia that any spontaneous increase in hemoglobin had occurred. Between March and September 1952 the hemoglobin ranged between 44 and 69% on blood transfusions every two weeks. The sedimentation rate was 56 on September 16, 1952. The real surprise came on September 30th when his hemoglobin was noted to be 80% and erythrocytes 4 million. Unfortunately, this improvement did not persist and more transfusions were subsequently necessary. However this was the highest recorded hemoglobin value since the onset of the anemia. The skin was now almost bronzed. Treatment was continued as before and he received two bottles of blood every two weeks but his hemoglobin remained at a higher level than it did formerly on the same quantity of blood. On June 25, 1955 the hemoglobin was 85%, the sedimentation rate 25

mm/hour. Transfusion was carried out as usual. This was the last blood transfusion given. The July 1953 follow-up showed a hemoglobin of 95% and he felt well.

He was not seen again until March 1954 when he returned because of hiccough. His hemoglobin was 100 per cent, erythrocytes 5.1 million and leucocytes 12,000, neutrophils 81, lymphocytes 18, monocytes 1, sedimentation rate 28 mm/hour, B.U.N. 11 mgs. per cent. Sternal marrow was reported as showing excessive iron present, but was otherwise normal. In May 1954 there was a recurrence of hiccoughs. Hemoglobin was 106%, leucocytes 12,000, neutrophils 94 and lymphocytes 6, sedimentation rate 18 mm/hour. In both cases the hiccoughs responded to anti-spasmodic therapy. Laboratory findings in June 1954 demonstrated the blood picture essentially unchanged, the fasting blood sugar 102 mgs. per cent, urine negative for sugar, and a mild pyuria which cleared after the passing of urethral sounds. Serum iron was 345 gamma % serum proteins 7.4 total, albumin 4.3, globulin 3.1 with alpha globulin 0.1, beta 1.3 and gamma fraction 1.7 grams per cent. His skin color was darker. There have been periodic examinations carried out in 1955 during which the blood picture has remained normal.

He was last seen on October 6, 1955. A glucose tolerance test showed fasting value of 100 mgs. %, 125 mgs. at the half hour, 110, 107 and 94 mgs.% at one, two and three hours respectively. Hemoglobin 103%, leucocytes 6,500, neutrophils 66, lymphocytes 31, eosinophils 3, sedimentation rate 8 mm/hour, hematocrit 43%. The serum proteins were about the same as in June 1954. The serum iron was 270 gamma per cent, thymol turbidity 3.5 thymol flocculation 4 plus and cephalin flocculation 4 plus. The B.S.P. retention was 29% in one half hour. Comparing these with the findings in January 1951 would indicate further impairment of liver function. There was no evidence of diabetes. The electrocardiogram records many premature beats and minor ischaemic changes. The skin was quite bronzed. He continued to feel well.

Comment

The etiology of refractory anemia presents a mystery as yet unsolved. The etiology of the hemosiderosis, however, is less mysterious, for the iron deposits in the tissues can be accounted for (at least partly) by the iron content of the transfused blood which, in this case, over a period of five years totalled about 40 grams, and to a lesser extent by ingested iron both medicinal and dietary. The latter two factors cannot, of course, be properly assessed, for even though absorption of iron is increased in anemia, the exact amount cannot be estimated in any individual case without painstaking balance studies.

Another interesting aspect of this case is the possible liver damage as suggested by the impaired liver function tests. Is the damage due to fibrosis secondary to deposition of iron, or is it a result of the anemia? These are questions which are difficult to answer.

While the medical philosophy of correcting one disease and inadvertently producing another poses a nice question, the situation cannot always be avoided, for both the physician and the surgeon encounter conditions in which undesirable effects appear in the course of life saving therapy.

Summary

A case of refractory anemia with spontaneous recovery, and a complicating transfusion hemochromatosis is reported and commented upon.

Acknowledgment

Due credit is given to those members of the Medical Service of Deer Lodge Hospital who over the years have also treated and recorded findings in this case, and acknowledgment made to the Laboratory and other services for their participation.

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Surgery

Carcinoid Tumors of the Gastrointestinal Tract

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Carcinoid tumors of the gastrointestinal tract present certain interesting characteristics which set them apart from other neoplasms, either benign or malignant, encountered in abdominal surgery. While carcinoids are not commonly encountered they cannot be classed as rare in the appendix, ileum, cecum, or rectum. Our interest has been stimulated by encountering three carcinoid tumors recently. After study of these cases and review of the literature we feel that certain pertinent points relative to their diagnosis and treatment are worth re-emphasis here. Any surgeon doing abdominal work should be prepared to recognize carcinoid tumors and deal with them upon the basis of those characteristics which set them apart from other tumors of the gastrointestinal tract.

Carcinoid tumours have been reported in the stomach, duodenum, ampulla of Vater, gall bladder, jejunum, ileum and Meckel's diverticulum, cecum appendix, colon, rectum and mesentery as well as in teratomas of the ovary in which they have usually been found arising from gastrointestinal remnants. The appendix is by far the most common site, accounting for about 70%[1] of reported carcinoids and occurring in 0.2 to 0.5% of appendectomy specimens. The ileum, cecum, and rectum are the next most common sites. Thus the gastrointestinal tract from the ileum to rectum accounts for the greatest bulk of reported carcinoid tumors, and the other sites mentioned fall into the category of rarities. This discussion will be limited to the more common sites mentioned.

Carcinoid tumors were first recognized as an entity in 1888[2]. The name carcinoid tumor has remained throughout the years despite objections by various authors that the name implies a benign tumor when actually it is now considered to invariably be malignant. Argentaffinoma has been the other popular name referring to the tumor cells affinity for silver stains. We shall use the name carcinoid since it still remains in popular usage. With the passage of years and the accumulation of more pathological knowledge it became apparent that while histologically relatively benign they frequently metastasize to regional lymph nodes and to the liver and not uncommonly distant hematogenous metastases are seen. For this reason the consensus of the present day opinion is that all carcinoid tumors are malignant although slowly growing, and late to metastasize. The metastases are frequently very slow growing and may even remain stationary for years. Terplan[3] reported a carcinoid of the cecum with hepatic and mesenteric metastases in which a palliative ileocolostomy was done; the patient died 3½ years later by drowning and an autopsy showed no progression of either the metastases or of the primary tumor. Merke[4] reported a similar case with death from pneumonia and erysipelas five years later and no progression at autopsy. Mallory[5] reported a case in which a segment of ileum was resected for a carcinoid and known mesenteric glands containing carcinoid were left behind. The patient died 20 years later of pneumonia and autopsy showed no further extension of the tumor. Two cases[13, 14] of small bowel carcinoid have been reported with survival for ten years following resection of the primary tumor leaving inoperable metastases. In other instances, and more commonly in rectal lesions, the metastases grow rapidly and the course follows that of other malignancies with early death although this rapid course is much less commonly reported than is the slow course. In general, larger carcinoids are more apt to metastasize but massive metastases from very small lesions have been reported. It must be emphasized that no criteria either gross or microscopic have been established to differentiate the slow growing or stationary carcinoids, with or without metastases, from those which spread and grow rapidly and kill quickly.

Whenever these tumors occur in the gastrointestinal tract they present certain gross characteristics common to all locations. They appear as firm nodules which are circumscribed, firm, submucosal and often are multiple. Two or more foci of tumor growth, of multiple carcinoids all considered to be primary rather than metastatic, are reported. The occurrence may be as high as 30% within the small bowel[6]. The tumor frequently projects into the lumen. While usually small they may reach 4.5 cm. in diameter and occasionally, particularly in the rectum, may be annular or polypoid and be grossly indistinguishable from adenocarcinomas. They are grayish-white to yellow and the cut surface is characteristically firm and yellow to light orange in color. The consensus of opinion now holds to the theory (amongst many) that carcinoids arise from the argentaffine cells deep in the crypts of Lieberkuhn. These cells are present throughout the gastrointestinal tract and the incidence of carcinoids roughly parallels the frequency with which these cells are found in the various parts of the tract. They are commonly associated with other malignancies of the gastrointestinal tract and this is particularly true in the colon where 31% of carcinoid tumors reported in one series[7] were

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associated with colon malignancies of the classical type. Microscopically, carcinoid tumors grow in solid sheets or cords. Very little anaplasia is seen and mitoses are rare. The cells contain chromaffin granules. These granules reduce ammoniacal solution of silver, staining black, hence the name Argentaffinoma. This reducing ability is one of the main criteria in establishing the diagnosis although some otherwise typical carcinoids do not take the silver stain and this is felt to be due to immaturity (pre-enterochrome phase) of the cells. Absence of silver staining does not mitigate against the diagnosis if the other criteria are present. In this regard it may be emphasized that carcinoid tumors may be passed over as classical adenocarcinomas and it is suggested that if any question arises as to the nature of a gastrointestinal neoplasm, silver stains and further pathological consultation and study be obtained before therapy is carried out.

Appendix

In the appendix, the typical carcinoid presents as a firm tumor usually in the tip of the appendix and is usually an incidental finding during appendectomy either for acute appendicitis or during routine incidental appendectomy. In the proximal appendix it may be obstructive and productive of a mucocele. Metastases from appendiceal carcinoids are found in less than 1%. This is probably accounted for by the slow growth of the tumor and the very small appendiceal lumen which produces pressure or obstructive symptoms early, thus resulting in the early removal of the appendix and tumor before metastases occur. Carcinoid, for this reason, should be suspected in any case of "chronic appendicitis". Whenever an appendectomy is done the possibility of a carcinoid should be kept in mind and if suspected can readily be confirmed by gross and frozen section examination. If the diagnosis is made in this manner careful search should be made for regional node involvement and hepatic metastases. If no nodes are found, wide resection of the mesoappendix should be done since direct extension into the mesoappendix is reported[6]. If nodes are found, radical resection to include right colectomy should be planned either immediately or as a second procedure after proper preparation. Thus in the presence of acute suppurative appendicitis one would not advocate the performance of a right colectomy. In the presence of an otherwise normal appendix, however, with positive nodes and good general condition of the patient one might contemplate immediate resection of the distal ileum and right colon with primary anastomosis. In general, a second operation, after adequate preparation, would be the procedure of choice. The low incidence of metastases should not lull the operator into complacency and thus cause him to miss the golden opportunity of curing a malignant lesion, which while slow growing is still a tumor which can metastasize and kill. In addition, the slow growing nature of the tumor makes a radical attack more likely to produce a cure in the presence of nodal metastases than would be the case in any other malignant neoplasm. If the carcinoid is unsuspected at the initial operation, or felt to have been completely removed, and microscopic sections show extension to or beyond the line of resection; a right hemi-colectomy must be done as a second operation without waiting for symptoms or signs of recurrence to appear. We would urge, therefore, that the possibility of carcinoid be kept in mind at the time any appendix is removed and the above mentioned course of treatment kept in mind. In addition, in those carcinoid tumors which are found by the pathologist after the completion of surgery, one may well keep in mind the second look principle of Wangensteen with the thought in mind of exploring and resecting metastatic nodes and even small metastases in the liver, if found. The very slow growing and even dormant nature of metastases makes such a plan feasible.

Ileum and Cecum.

The ileum and cecum are the next most common sites and may be considered together since any radical surgical procedure for either lesion must be the same. Thus, a carcinoid involving the ileum, cecum or ascending colon would require a right ileo-colectomy in order to remove the tumor and adequately remove the regional nodes. Carcinoids account for 23% of malignant small bowel tumors[12]. Metastases occur in 25[9] to 38[10]% of these lesions. The symptoms are essentially those found with other malignant tumors in the same area. Small bowel obstruction is usually due to adherent loops of bowel from direct serosal extension of the carcinoid, or an associated inflammatory reaction, rather than to stenosis or intraluminal obstruction of the bowel[6]. Again it is important to recognize the tumor at the time of operation. We consider this particularly important since the slow growing, but clearly malignant nature of carcinoids makes radical attack upon such a tumor practical and feasible when a grossly similar other type of malignant tumor with nodal metastases would clearly indicate a simple palliative resection, since such a malignant lesion would obviously be beyond hope of cure by any more radical procedure. An obstructing carcinoid of the terminal ileum with multiple mesenteric nodal metastases might well be considered hopeless, and a local resection of the obstructive area done palliatively if carcinoid was mistaken, (and it easily can be) for an adenocarcinoma. On the other hand, if carcinoid is kept in mind, suspected and then diagnosed by frozen section, a more radical attack could be undertaken with a much

greater chance of cure or many years of palliation. In the same vein one can even postulate that in the presence of one or two palpable hepatic metastases and a locally resectable lesion with or without nodal involvement a radical resection including all available nodes should be done. A second operation could then be planned for local resection of hepatic metastases if surgically feasible. Once again the slow growing characteristics usually manifested by carcinoids of the small bowel makes such surgical thinking feasible where it would not be with any other malignant tumor of the bowel. For these reasons recognition of carcinoid tumors at the time of operation seems to us to be important if the best opportunity for cure is to be grasped. In the same light, the principle of "the second look" operation as advocated by Wangensteen, should be particularly applicable when dealing with carcinoid tumors.

The remainder of the colon with the exception of cecum and rectum is rarely the site of carcinoid tumor growth. If found in the transverse or descending colon the treatment should be resection as for any other malignant tumor.

Rectum.

The third most common site is the rectum where the tumor has the previously described gross appearance, but may ulcerate, become large and fungating and thus grossly be indistinguishable from the common malignant tumors encountered therein. A large percentage of rectal carcinoids occur on the anterior wall 5 to 10 cm. above the anus. In one series[8] 64% were found in this area. A high percentage of rectal carcinoids will therefore be within reach of the examining finger and serves to reemphasize the importance of routine rectal examination. Here we would again suggest somewhat different surgical thinking than that applicable to the common carcinomas encountered. It may be urged that carcinoids be kept in mind whenever an atypical tumor is found on biopsy. Several combined abdominoperineal resections have been reported[12] and many others undoubtedly have been done for very small rectal carcinoids which could undoubtedly have been cured by local or anterior resection if the tumor had been recognized as an entity prior to operation. As previously mentioned, if the diagnosis is not clear to the pathologist on the initial section, silver stains, further biopsy and additional pathological consultation may be in order since the treatment may be modified if carcinoid is recognized before definitive surgery is performed. Having recognized a carcinoid of the rectum we would urge the following plan of action. A small localized carcinoid may be treated by wide local excision particularly if it is pedunculated. If microscopic study reveals a good margin of excision, this may be all that is necessary to cure. Re-operation should be considered with abdominal exploration as for more advanced lesions if microscopic study indicates an inadequate margin of excision or if there is any sign of recurrence or suspicion of nodal involvement. A larger lesion or multiple lesions, particularly if there is ulceration or obvious infiltration, should be treated by abdominal exploration and radical resection. Because of the nature of carcinoids, however, we feel that the criteria for anterior resection may be extended, particularly in relatively early or localized lesions, so that the sphincter may be preserved more often than is safe or feasible in the ordinary rectal malignancies. When the rectal carcinoid is more advanced and fungating, or annular and infiltrating widely, combined abdominoperineal resection, or anterior resection with the same criteria for choice of operation, as for other malignancies should be considered. If there are multiple liver metastases the criteria for low anterior palliative resection may again be extended in order to rid the patient of the fungating tumor, and with the hope at least, for many months of comfort.

In summary then, carcinoid tumors are a distinct entity in themselves. They occur most commonly in the appendix, ileocecal area and rectum, in that order. In the other sites mentioned they are rare. Most authorities now consider all carcinoid tumors as true malignant growths. They are usually slow growing and in a number of cases apparent arrest for years has been reported. In the light of these characteristics, which set carcinoid tumors apart from other malignant tumors of the gastrointestinal tract, it is suggested that the diagnosis of carcinoid be kept in mind whenever dealing with an atypical tumor of the gastrointestinal tract in order that a somewhat different surgical approach to the problem may be undertaken.

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SERIES IX

Aetiology and Research in Cerebral Palsy

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The attached table is a break down of the possible aetiological factors in 259 cases of cerebral palsy examined, under the auspices of the Society for Crippled Children of Manitoba during the years 1950-54, by members of the staff of the Winnipeg Children's Hospital. Of necessity reliance had to be placed on the history as given by the parents. Time has not allowed for examination of hospital records in every case. Unfortunately such records frequently leave much to be desired.

Where our own records were inadequate, or in those cases, after questioning the Mother closely, in which there seemed inadequate aetiological factors, the case was added to the unknown column. Anoxia was used for those cases in which labour was prolonged or difficult, and preferably where the child was known to be cyanosed at birth. Trauma was used where precipitate delivery, difficult delivery or Caesarean were the main factors; also in those cases that had convulsive or other signs suggesting intra-cranial haemorrhage at birth. Obviously the division between anoxia and "trauma" on past history is not easy. The acquired group include an onset after infancy due to meningitis, trauma and other causes. The congenital and familial group contain cases in which more than one child in the same family was afflicted with the same lesion. For the sake of simplicity the occasional triplegia was included in the quadriplegic group. Quadriplegia denotes involvement of all four limbs, usually asymmetrically. Again paraplegia and diplegia are grouped together as indicating a predominant symmetrical involvement of the legs. The two monoplegias in this series are included under hemiplegia.

When comparison is made between the various groups, the "unknown" group prove to be a useful control series for prematurity. Perlstein* in his paper on clinical correlates postulates that diplegia is most commonly associated with prematurity. To a certain extent this is true in our series. However, the more important observation to be gleaned from this series is the striking similarity in numbers, with the possible exception of diplegia, between the various lesions in which the aetiology was either prematurity or unknown. This would suggest that prematurity per se may bear some relationship to diplegia, but is not the only factor involved in the children with quadriplegia, athetoid or hemiplegia. Comparisons between other groups can also be made. For instance a fair proportion of quadriplegics arise in the congenital and familial group. Hemiplegics are noticeable under trauma and acquired. Athetoids predominate in the anoxic and rhesus groups. Is it, in fact, anoxia, or a raised serum bilirubin, that accounts for athetosis following premature delivery? Or trauma, or genetic factors, accounting for hemiplegics and quadriplegics respectively?

It would seem that, insofar as the premature group represent 25% of the aetiological factors in cerebral palsy, it would be the most profitable section for further research. Moreover such a study must be a forward one rather than in retrospect. A good premature unit with one man making detailed records of all the possible antenatal and natal factors at the time of delivery is the only accurate method of approach. At the same time repeated examination of these children whilst in the nursery might provide additional information on the early signs of cerebral palsy. This would be a long term project, as follow up of these children would be necessary to confirm the presence or absence of cerebral palsy. Many hundreds of premature births would have to be followed up if the most conservative figures of incidence of cerebral palsy were taken to be 1 per 1000 births. The highest figure given is 1 per 200 births. Studying a premature group only, the incidence would be expected to come nearer the highest incidence.

Concurrently more research could be done in the Province of Manitoba on the figures becoming available. No statement has been published regarding the incidence of cerebral palsy in Canada. There is considerable variance between figures given by the British and Scandinavian writers on the one hand, and those of the United States on the other. Preliminary estimates suggest that in Manitoba our figure will be in line with the former i.e. 1 per 1000 births, or 3 - 4 born annually per 100,000 population, giving a total under 21 yrs. of 450-500 cases. Of this estimate, over 300 cases have been registered to date in Manitoba. Apart from an active case finding programme, considerable help could be obtained, particularly in finding the minimally disabled, if all doctors in the province would voluntarily notify such cases to the Central Registry for the Disabled.

*M. A. Perlstein, J.A.M.A., 149: 30, 1952.

Further work on the present figures is required in regard to obtaining more accurate records of aetiology from hospital records, family physicians and the parents themselves. In the premature group more detailed analysis can be done in regard to sex, age, birth-weight and place of birth. Such a continuing retrospective study would help the forward study and both could run concurrently.

Another project would be detailed neurological assessment of the quadriplegic group. Some of the older clinicians have used the term bilateral hemiplegia to describe a distinctive type of quadriplegia in which the two sides of the body are asymmetrically effected. Moreover the arms are usually more severely afflicted than the legs. The object of enquiry would be to try to distinguish this bilateral hemiplegia from other types of quadriplegia. Finally, if such a distinction were found possible, an attempt would be made to correlate these cases with hemiplegia and see if the same aetiological factors occur.

Conclusion

The crucial problem in cerebral palsy does not lie in costly treatment centres, it lies primarily in prevention. Already it has been recognized that if the serum bilirubin is kept below 20 mgms% in cases of rhesus incompatibility, Kernicterus can be prevented. Research in Manitoba is already in progress on the relationship between the serum bilirubin level and subsequent brain damage. The incidence of cerebral palsy will automatically fall with improved antenatal and natal care. Cases associated with breech and Caesarian deliveries, with so-called trauma and anoxia can be reduced as technique advances. Finally improved premature care will obviously show good returns. Many factors in aetiology remain obscure, and it is hoped that by working along the lines suggested in this paper, that they can be elucidated, and prevented, to the ultimate benefit of humanity.

1. Premature— 69 (26%), 14 Quads, 24 Diplegic, 19 Hemiplegics, 9 Athetoid, 3 Ataxia.
2. Unknown— 62 (24%), 16 Quads, 12 Diplegic, 21 Hemiplegic, 12 Athetoid, 1 Ataxia.
3. Anoxia— 31 (12%), 4 Quads, 2 Diplegic, 10 Hemiplegic, 14 Athetoid, 1 Ataxia.
4. Acquired— 28 (11%), 6 Quads, 1 Diplegic, 19 Hemiplegic, 2 Athetoid.
5. Trauma— 25 (10%), 5 Quads, 2 Diplegic, 13 Hemiplegic, 3 Athetoid, 2 Ataxia.
6. Congen. and Familial— 16 (6%), 9 Quads, 2 Paraplegic, 3 Hemiplegic, 2 Friedreich's Ataxia.
7. Rhesus Incompatibility— 15 (6%), 14 Athetoid, 1 Ataxia.
8. Breech— 7 (3%), 2 Quads, 1 Paraplegia, 4 Hemiplegia.
9. Toxaemia— 6 (2%), 1 Quad, 4 Hemiplegia, 1 Athetoid.

Otolaryngology

Deafness in the Pre-School Child

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Introduction:

It has been estimated that between 3-4% of the school children in Winnipeg have some degree of hearing loss requiring some form of treatment. The purpose of this article is to stress the importance of diagnosing and treating the hard of hearing child even before he becomes of school age. There are now available new and adequate methods of testing the hearing function in children this young. We can no longer accept as good medical practice the old fashioned advice to the mother "bring the child back for a hearing test when he is old enough to co-operate." In his first few years a child learns speech mainly through adequate hearing and imitation of spoken voice. Between the ages of two and five years he is at his peak as a language-learning individual; never again will he exhibit such readiness, and eagerness for the acquisition of language and speech.

Etiology:

The causes of deafness are many. They must all be considered if we are to make an accurate diagnosis which in the end determines the successful management of the case. Detailed history and thorough examination are of prime importance.

(a) Pre-Natal Causes:

(1) During preconception: syphilis, hereditary factors (e.g., otosclerosis, inherited familial deafness.)

(2) During pregnancy: 1. Malformations (e.g., atresia ear canal). 2. Viral infections during the first trimester (e.g., rubella). 3. Erythroblastosis fetalis (if inadequately treated so that a significant degree of jaundice persists in the first 2-3 days of life). 4. Excessive use of toxic drugs (e.g., Quinine).

(3) During labour: 1. Birth trauma. 2. Otitis neonatorum.

(b) Post-Natal Causes:

(1) Otitis media: usually associated with eustachian tube disease causing obstruction.

(2) Infectious diseases: meningitis, encephalitis, etc.

(3) Poisons: (e.g., excessive doses of, or sensitivity to dihydrostreptomycin, salicylates, quinine.)

(4) Miscellaneous: cerumen, leukemia, cerebral degeneration, aggravation due to psychic factors, trauma to auditory pathways, etc.

Investigation and Diagnosis of Deafness:

This cannot be accomplished by the otolaryngologist alone. Team work is essential to arrive at the correct diagnosis. The parents often first suspect or realize that the child has a hearing defect because of poorly developed speech or lack of response to spoken voice. The general practitioner may be consulted or the child referred to a pediatrician. A thorough physical examination and history may reveal the probable cause. The otolaryngologist must exclude disease in the ears, nose, nasopharynx, sinuses and throat. The child psychiatrist may be called on to assess the adequacy of development and to consider the importance of any emotional factors or problems present.

Hearing function is tested by the audiologist. The procedures used in adults and older children i.e. speech testing, pure tone audiometry and tuning fork testing, are not satisfactory for the preschool child.

There are three types of equipment which are being used for the testing of the infant and preschool child:

(1) Peep show audiometer
(2) Psychogalvanometer
(3) Electroencephalogram

The first two tests are generally used in children between three and six years and are based on the principle of the conditioned reflex. The child is conditioned to sound by light (in peep-show audiometry) and mild electrical skin shock (in psychogalvanometry). A description of the actual test and method of testing is beyond the scope of this paper. It has been our experience in common with many others, that the hearing capacity in a child can be accurately determined by these methods. Patience and understanding are necessary here as they are in all procedures having to do with small children. Audiometry with the electroencephalograph is likely to be the preferred method with infants and very young children. Sounds of different intensity and frequency are introduced to the patient's ears at the same time the E.E.G. tracing is being taken. A characteristic change in the graph pattern will be noted when the child hears. Other procedures have been used, such as calibrated noise-makers and special recordings of sound at different levels, but the results have not been consistent and dependable. The peep-show method appears to be the most acceptable to the child from 3-6 years and the conclusions have been quite consistent in our hands.

Management and Recommendations:

Treatment and training should be started as early as possible in the child's life. After investigation and diagnosis comes treatment. This of necessity depends on the cause of the deafness and its severity. Any existing infection in the ears, nose, sinuses or throat must be treated. Tonsil and adenoid surgery may be indicated. Excessive lymphoid hyperplasia in the nasopharynx blocking the eustachian tube may be treated by radiation in the form of X-ray or a radium applicator. This is of great importance as there may be lymphoid tissue in the eustachian orifice which cannot be satisfactorily removed surgically. Eustachian tube inflation or politzerisation may be necessary to open an obstructed or partially closed tube. In cases of serious otitis media with fluid accumulation in the middle ear, aspiration of the fluid through the ear drum or myringotomy and spot suction may be advisable. Atresia of the external auditory canal will require surgical correction. Mastoid operations may be necessary in carefully selected cases of otitis media and mastoiditis especially if associated with polyp granulations and cholesteatoma formation. Patients with otosclerosis who still have good nerve conduction can be helped by the fenestration operation.

The above conditions produce primarily a conductive type of deafness. The prognosis with treatment in this group is good—much can be done to bring the hearing back to within normal limits.

A second group of patients suffer from nerve deafness. Here the damage is usually of a permanent type and the prognosis for restoration of normal hearing is poor. In this group we see patients with deafness following rubella, meningitis, cerebral damage, poisons, etc. The rehabilitation will depend on the severity of deafness. Favourable classroom seating, lip reading, hearing aids and special speech and hearing instruction may be necessary. In complete or near total deafness a special school for the deaf child may be required. Speech can be taught to children with the aid of amplified sound, provided the diagnosis of deafness and the training are started early in life. A great deal of patience and understanding is essential.

Case Reports

Case 1

J. B. is an eight year old girl with bilateral hearing loss for the past few years, following repeated ear infections and recurrent tonsillitis. A tonsillectomy and adenoidectomy was done in 1952 following which there were no further ear infections. The hearing, however, did not improve. Examination in December 1954 revealed excess lymphoid hyperplasia in the nasopharynx which was blocking the eustachian tubes. Hearing tests showed a conductive deafness, and the ear drums were retracted. A diagnosis of eustachian

tube obstruction due to lymphoid hypertrophy was made and the patient referred for radiation treatment to the eustachian orifice regions. A repeat hearing test two months later indicated that the hearing was now within normal limits.

This case shows the conductive type deafness seen with disease of the middle or external ear and the eustachian tube. The hearing loss is due to blockade of or interference with the sound conducting apparatus. The treatment is usually satisfactory and the prognosis good as the hearing can be restored in the earlier stages.

Case 2

J. W. is a seven year old boy with hearing and speech disorders. The patient had severe jaundice due to erythroblastosis fetalis and was treated according to methods in vogue in 1948. The patient's two older brothers had no history of jaundice at birth and both have normal hearing and speech. The deafness was obvious since infancy. Hearing tests revealed a marked bilateral nerve deafness. The ears appeared normal on examination. Hearing and speech rehabilitation with the use of a hearing aid were started at once.

This is a case of congenital nerve deafness. The disturbance is in the central auditory pathway and is likely due to pigmentation deposits in the auditory tracts and nuclei. The deafness is irreversible. However, with rehabilitation, lip reading, and amplified sound (hearing aid) useful hearing and speech may be attained. This type of deafness should become much more rare now that exchange transfusions are carried out almost at the moment of birth in babies with jaundice from erythroblastosis.

Case 3

T. R. is a six year old boy with deafness and poor speech development apparent since birth. The family history is remarkable in that both parents were born deaf; the mother's brother and sister were born deaf; and the patient's brother and sister are deaf mutes. Ear, nose and throat examination revealed no abnormalities and hearing tests showed a severe bilateral nerve deafness.

This is a case of congenital hereditary nerve deafness. Rehabilitation plan is similar to that for Case 2.

Conclusion

(1) Early diagnosis of hearing loss can be established with certainty in the preschool child by the use of newer diagnostic methods.

(2) Early diagnosis makes early treatment possible.

(3) Early diagnosis and prompt treatment will result in more children entering school-life with little or no hearing and speech handicap.

(4) The mental and physical well-being of the child resulting from correct diagnosis, and rehabilitation or cure, will make for a happy childhood and a successful school career.

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Abstracts from the Literature

Huntington's Chorea: Results of Treatment with Reserpine. J. A. Lazarte, M. C. Petersen, C. W. Baars, J. S. Pearson. Staff Proc. of Mayo Clinic, 30: 358, 1955 (Aug 10).

19 of 21 patients with Huntington's Chorea were treated for an average of 70 days with Reserpine. The initial dose was 5 mg. of reserpine by I.V., with 4 mg. by mouth, for each of three days. Then an oral maintenance dose of 4-11 mg. per day was used. Hyperkinesis was decreased markedly in the majority of patients. Impaired voluntary motor function was improved. Mental disturbances were least influenced by treatment. The side effect of inertia was seen and controlled. Reserpine in the treatment of Huntington's Chorea is regarded as purely symptomatic. It might be used to attempt to reduce hyperkinesis in chorea minor, chorea gravidarum, athetosis, ballism, torticollis, hepatolenticular degeneration, dystonia, tics, spasms, and tremors.

A. G. Rogers.

Occult Carcinoma of the Prostate. L. F. Greene, H. V. Simon, J.A.M.A., 158: 1494, 1955 (Aug. 27)

83 cases of occult carcinoma of the prostate were found in a review of some 2,000 cases of cancer of the prostate. These patients were treated by transurethral prostatic resection. The survival in 5 years was 95%, and in 10 years 85% of the expected survival rates for the normal population of this age group. The mortality and morbidity of occult prostatic cancer is low. Because of this, the advisability of radical prostatectomy in occult carcinoma of the prostate is questioned.

A. G. Rogers.

Pathology

Cancer and the Cell

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Since this meeting is described as a clinical luncheon, I felt sure I should present material with some immediate clinical application. However, during a few years preceding my arrival in Canada I had been engaged on experimental cancer research, therefore I felt I would be happier if I were allowed to speak on this subject. The type of work I was doing was sometimes described, perhaps even with a hint of opprobrium, as "ivory tower" work. Francis Bacon once said that anyone who lived in an ivory tower must have a head composed of the same substance. So, since this is a clinical luncheon I shall try, in the course of my address, to show how experimental cancer research is directed towards the solution of human cancer problems.

Cancer research can be divided into two main categories. The first is clinico-pathological. This consists of the continual attempt of hospital pathologists and clinicians to correlate the macroscopic and microscopic appearance of tumours with their behaviour in human beings. Unfortunately this branch of research is faced with an almost insuperable obstacle. The obstacle is the patient—consideration of the patient's welfare has a bad effect on the scientific detachment of the pathologist. Any pathologist can diagnose the obviously malignant or obviously benign tumour. But there are many instances where the malignancy of a tumour is in doubt. Consciously or unconsciously the pathologist, with the patient's welfare in mind, tends to err on the side of caution and thereby may put the label of cancer on something which is, in fact, not cancer. This difficulty was pithily put by a distinguished German pathologist while looking at a histological section of irregular endometrium. He said, "This is not cancer, but the uterus had better come out." This is the sort of thing which may account for the relatively favourable prognosis of carcinoma of the body of the uterus.

In 1936 a small skin tumour called molluscum sebaceum was described that was self healing and benign. It was forgotten until about a year or two ago when interest in it was revived. It was found that this lesion was being diagnosed as grade I carcinoma of the skin and these lesions in one hospital comprised about 30% of all skin cancers. If these skin lesions are really not cancers, and there is good reason to believe this, then our cancer statistics are surely being distorted.

Presented at the Clinical Luncheon, Municipal Hospitals, Winnipeg, Man.

Nowadays the poor hospital pathologist is being pushed to even greater extremes—he is being asked to diagnose cancer even before it has arisen. He is being asked to diagnose carcinoma-in-situ of the cervix. From an examination of the literature it is significant that there is no general agreement on the exact histological criteria of carcinoma-in-situ of the cervix. Nor is it obvious that invasive carcinoma invariably or even usually follows so-called carcinoma-in-situ. It is a great pity that the label of cancer with all its sinister connotation was ever applied to this lesion. It forces the pathologist to give the name cancer to something which may well not be cancer. The result of this may cause patients to suffer unnecessary and acute distress.

In view of such difficulties I think it is obvious that opportunity should be given for research to be carried out, if not in an ivory tower, at least well removed from the confusion introduced by the patient and from the importunities of clinical colleagues.

At present we have no precise definition of cancer—its fundamental characteristics are unknown. We cannot tell for certain by the appearance of a single cell whether it is a cancer cell or not. If we can find some method of doing this even at the cost of supporting many researchers full-time for many years, it would be a tremendous advance against a disease which kills people at an age when they are of most value to the community.

This brings me to the second of the two major categories of cancer research—that is to say experimental cancer research.

Experimental cancer research depends on the fact that the analogues of human tumours occur in animals such as mice, rats and rabbits. These tumours do not differ in behaviour and appearance from those occurring in human beings. This branch of research can be said to have begun in 1890 with the discovery that it was possible to remove such tumours and introduce pieces of them under the skin or elsewhere in other animals where they would grow. Such tumours are known as transplantable tumours, and their behaviour under many different conditions has been studied all over the world.

Transplantable tumours have an obvious application, which has been well exploited, and that is in the testing of anti-cancer agents, such as chemicals, x-ray and radar waves. In fact practically anything you can think has been tried including extracts of herbs gathered from the Welsh hills by the light of the full moon and extracts of horses' urine.

Many investigations have been designed to discover what physiological and biochemical changes take place in animals bearing tumours. Some of these investigations have been carried out in order to try and devise a test or tests for cancer. One such piece of work is that of McHenry, Professor of Nutrition at Toronto and his associates. They applied their experimental findings to human beings and found that glutamic acid was formed in the blood in greater concentrations than normal in people with malignant growths. This was not found to be so in people with benign growths nor in pregnancy. This piece of work, which arose from laboratory experiments, does not seem to have received the attention it deserves.

The effect of the administration of hormones and the excision of the various ductless glands on animals bearing transplantable tumours is being studied. Such work is extremely pertinent at the present time when cancer in humans is being treated with hormones and by ablation of the gonads, adrenals and pituitary glands.

All the experimental work that I have described so far is designed to be immediately applicable to present day clinical problems and can therefore be described as applied experimental cancer research. Fundamental cancer research is designed to discover the nature of cancer itself—how cancer cells differ from normal cells. There are four main aproaches.

1. Heredity
2. Viruses and cancer
3. Chemical carcinogenesis
4. Cytology, or the study of the minute structure of the cell.

It was originally thought that cancer was hereditary because it was observed more commonly in some families than others. This applied to both humans and mice. Experimental work on the heredity of cancer in mice began when a young girl named Maud Slye literally burst into the laboratory of a pathologist in Chicago and said she was convinced that cancer was hereditary. She begged for a corner in the laboratory and for a few mice. The few mice grew into thousands and the corner of the laboratory into an institute. Maud Slye was the leader in this field for well over ten years with many publications to her credit. She believed that cancer in mice was due to a recessive gene. Unfortunately her zeal and partisanship for her preconceived idea clouded her scientific judgment. And, at a scientific meeting, which is reported in full in the now extinct American Journal of Cancer, she was subjected to a harsh attack by some dry as dust statistician, and her ten years or more of work was apparently set at nought. After this, as far as I could determine, she published only one more paper and quietly faded from the scene. You may say that here is an example of wasted time and money—but I don't think so. I think that Maud Slye's work, faulty as it may have been, stimulated a great deal of experimental work on the influence of heredity on cancer. Also it was the stimulus which resulted in the discovery of the milk factor, which I will refer to later.

Breeding experiments have been carried on for many years now. Strains of mice have been developed in which the percentage incidence of cancer of a particular organ can be predicted with great accuracy. There is no doubt that heredity can, under these admittedly highly artificial conditions, influence the incidence of cancer. It has taken about 10 years to achieve these results. If one were able to control the breeding of a group of human beings it has been calculated that it would take 800 years to achieve the same results as in mice. However, there is some evidence that the offspring of human families with cancer are likely to have a slightly increased chance of dying of the disease. For practical purposes however, the increased chance is so small as to make no difference to individuals. Also, of course, human beings are protected from heredity cancer by their haphazard breeding habits.

That viruses cause cancer was put forward by Borrel in the last century on purely theoretical grounds. But at the turn of the century an Italian, Ciuffo, managed to transmit some warts from one of his patients to himself with a saline cell-free extract of the warts. In the laboratory in 1908 Ellerman and Bang transmitted a chicken leukemia in the same way. This was followed in 1914 by the discovery of Rous of his famous sarcoma in Plymouth Rock chickens. This tumour was also transmitted by cell-free filtrates. Next, Shope investigated the cottontail rabbits of Iowa and Kansas that were subject to papillomatosis of the skin. Shope found that these papillomata could be transmitted by cell-free filtrates. In each of these examples the active agent has been shown to be a particulate body by electron microscopy, filtration experiments and analytical centrifugation. In short the active agents have the fundamental properties of viruses.

Bittner, whose work arose out of that of Maud Slye, bred some strains of mice which had a high incidence of breast cancer. However, the results obtained with these strains did not fit into the Mendelian framework of heredity. He decided that there must exist a factor outside the chromosomes —the extrachromosomal factor. As a result he performed Caesarian sections on high cancer strain females and allowed the young to be suckled by a female of low cancer strain and vice versa. The young mice suckled by the low cancer strain female had a low incidence of breast cancer and those suckled by the high cancer strain female had a high incidence. He concluded naturally enough that the extrachromosomal factor must

reside in the maternal milk and he called it the milk factor. This work has been amply confirmed and electron microscopy has revealed virus-like particles in the milk of high cancer strain mice. These finding have been applied to human beings and a report from the United States on the electron microscopy of human milk has shown similar particles to be more numerous in women who have a high incidence of breast cancer in their families. A somewhat similar report has appeared in Britain. However, it is a relief to know that, among mice, only certain strains are susceptible to the effect of the milk factor. As has been indicated before, such pure strains are unlikely to occur among human beings. To summarize the work, it is undoubtedly true that some animal cancers are caused by viruses and it is possible that some human cancers are too.

The first observation which started the investigation of chemical carcinogenesis is well known. It was made in 1775 by a London physician, Percival Pott by name. He noticed that cancer of the scrotum developed in young men who, when they were boys, had been sent up the large chimneys, common in England at that time, to clean soot from them. The soot permeated their clothes and together with the constant irritation in the region of the scrotum caused ulceration and cancer. It was not until 1915, although previous attempts had been made, that the patience and persistence of two Japanese, Yamagiwa and Ichikawa produced cancer of a rabbit's ear after prolonged application of tar. The next step was when Kennaway and his co-workers started in 1921 to try and isolate the active chemicals in the tar. It was not until 1933 that his devotion to the task produced the successful final result. Now, there are available many chemicals of which are known to be particularly potent carcinogens. Fundamental work in this field has reached a point of refinement where the structure of chemical carcinogens is being related to the chemical structure of the component parts of the cell. We are all familiar with the application of this work. As a result of it, it is now known from laboratory tests on mice that 500 trade-catagories in the United States are potentially dangerous owing to the use of carcinogenic chemicals. The results in this field are helping to build up a case against tobacco as carcinogen and as the cause of lung cancer. And, you may be sure that the cigarette manufacturers are busily trying to produce carcinogen free tobacco. What more practical application of experimental work could you want?

Whatever the exciting cause of cancer may be—carcinogenic chemical, virus or hereditary disorder or a combination of them, the ultimate change characteristic of cancer must reside within the cell. It is not anything outside the cell being produced by the body because we know we can transplant a cancer cell from one animal and it will grow equally well in another. We know that the causal agent in the cell is not large or it would have been seen under the light microscope. We are also fairly certain that it is not a simple chemical compound — otherwise it would have been discovered by now. Therefore all that is left is a chemically complex particle of the same order of size as a virus or large protein molecule. So research into the minute structure of the cell is being pursued zealously by such methods as electron microscopy and cytochemistry.

The difficulty in this field has been to obtain single cancer cells. If one uses solid tumours one has to employ the relatively crude methods of histology or of mincing the tumours finely to try and obtain single cells. It is possible to grow tumours in tissue cultures. These have one great drawback for many purposes, which is that the cells will grow only on a surface such as glass or cellophane or in a plasma coagulum. This is convenient enough for morphological studies, but it does not allow one to handle the cells— to count them in a counting chamber or to titrate them as one would titrate viruses. A method of obtaining a disperse suspension of tumour cells has been badly needed, not only for fundamental work but also for quantitative measurements of the effect of anti-cancer agents. It is required so that we can test chemical against cancer cells in the same way as penicillin is tested against bacteria.

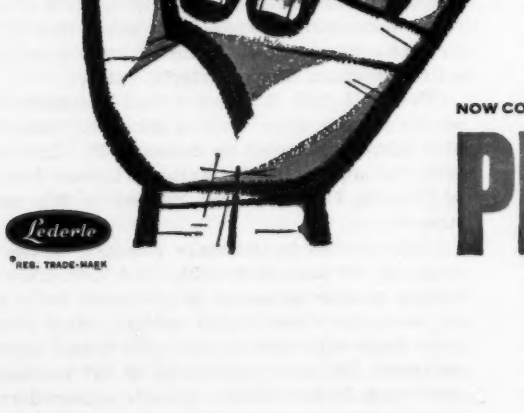
With such thoughts in mind, in 1951, interest was revived in the so-called ascites tumours described several years before. It was found that if an emulsion of cells from a subcutaneous tumour were inoculated into the peritoneal cavity, an ascites would develop which contained single cancer cells. These cells would increase in number until the death of the animal. The fluid could be inoculated into other animals where it would produce ascites again. This finding was greeted with enthusiasm— the thought being that at last we had a pure culture of cancer cells growing in a fluid medium, like a bacterial culture.

Before I left England I had established an ascites tumour from a subcutaneously transplantable neoplasm known as Sarcoma 37. Last year, with the support of the National Cancer Institute of Canada, I resumed my interest in this ascites tumour.

I discovered in the early passages, in the late stage of the tumour growth, that invariably soft diffuse tumour appeared in peritoneal folds lying between the stomach and spleen. Also I found very frequently that tumour cells would diffusely infiltrate the small collections of fat enclosed in peritoneal folds. Similar growth appeared in the bodies of fat attached to the gonads of the mouse. This infiltration could be demonstrated before

there was any increase of sarcoma cells or of fluid in the peritoneal cavity. At a later stage I could demonstrate growth of sarcoma in thin vascularized layers over wide areas of peritoneum. Even when ascites alone was present and no tumour visible to the naked eye, there was growth in the interstices of the fat and on the surface of the peritoneum.

The conclusions I have drawn from these experiments is that the ascites tumour develops by sarcoma cells finding their way to places where there are blood vessels. Once near blood vessels they multiply. As they grow further away from the source of their nutriment, they separate and pass into the peritoneal cavity. I believe too, that multiplication does not take place in the fluid at a distance from the nourishing blood vessel. If this is so then the cells in suspension in peritoneal fluid are dormant although they are still viable. Admittedly I have not proved all my explanation, but I have shown that it is possible, and there are good theoretical reasons for believing it probable. If I am right it is of importance because many workers are using ascites tumours for chemical and growth investigations. They are assuming that they are dealing with actively multiplying cells, when in reality they may be dealing with dormant cells.



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Tuberculosis

Processing Positive Survey X-ray Chest Films

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Public health or community health is only the summation of individual health. The broader or collective problem can only be handled in so far as the isolated or personal one is cared for. It is true that the two problems are inseparable, and one can only be effectively handled by considering the other. Personal health is primarily the responsibility of the practicing physician, and must of necessity be handled on an individualized basis. Practicing physicians, of course, include not only private practitioners but also those treating patients in institutions. Community health is the responsibility of many individuals and groups of persons, official and voluntary. But the practicing physician is a most important member of such a group or team. He is the one who ultimately takes charge of the patient.

To best utilize the talents and abilities of the practicing physician in the public health team one must have direction. The comprehensive broad plans of prevention and control must be explored and mapped out by officials trained and capable in that field. The team must be organized, but in that planning the practicing physician must be not only consulted, but included. He is willing and capable to work as a part of an organized effort and under direction when needed. The public health official or agency plans for mass action, be it X-ray surveys, group tuberculin testing, collective B.C.G. vaccination, community immunization or general educational campaigns. The ultimate objective of all such actions is the individual, and in the actual application of prevention or cure to that individual the practicing physician is responsible.

An abnormal X-ray chest film found in a survey is an objective symptom presented by the physician to the patient. This finding may mean nothing to the health of the patient. It may mean much. Having once inaugurated this complaint for the patient, it behooves the physician to explain it. The psychological response of the afflicted person may vary tremendously. You may start a reaction of fear out of proportion to the apparent threat of health, a fear that is hard to handle. Or you may have to plead, coax, and threaten to force the patient to pay proper attention to the findings. Your first approach to the person involved is extremely important and delicate because you are initiating a complaint for the person and you do not know how serious or how inconsequential it may be. Therefore, approach the discussion with judicious and tactful acknowledgment of not knowing the answer and yet with firmness that assures the person that it is to his best interest to work with you to find the answer. The full benefit of the medical finding can be made or lost to the person by this initial discussion with the physician. It is my judgment that on the whole the private physician has done a creditable job in this respect.

The X-ray chest survey can prove of big value to the individual patient but also can stimulate widespread interest in individual and public health by the general population. The survey also serves to stimulate the doctor, whether he be general practitioner or specialist. In using the X-ray chest survey as a tool of education for the public we must beware of engendering fear. We must guard against jumping at first conclusions and diagnoses from the study of the survey film. This marks only the beginning of a study both for the doctor and patient.

Organized educational and promotional campaign has fallen short of desired response goals among the older age groups. Since the preponderance of deaths from both lung cancer and pulmonary tuberculosis fall among the older group, this could, perhaps, be taken as a serious shortcoming of the mass radiographic approach to case finding. In this we are today paying for concentrating in past years on segmental case finding in tuberculosis. The group most often screened in earlier years, industrial population, student groups and military recruits, were largely those in the younger age groups. Unwittingly we have, perhaps, permitted our older population to come to a false sense of security about tuberculosis because of our failure to include them in these early programs. This situation is now being corrected. Moreover, the fact that X-ray surveys today attract a greater proportion of the younger population does not represent as great a loss as may first be thought. The health education and periodic X-ray habit formation, by products of a screening survey, are bound to be reflected in future surveys and health habits of individuals. Today mass X-ray screening programs are being conducted at a volume sufficient to make them very productive avenues of case detection of chest diseases. Small film chest X-ray surveys by official and voluntary health agencies in the U.S. have reached a level of over fifteen million examinations per year. An earlier feeling of reluctance and actual antagonism to X-ray chest surveys on the part of practicing physicians, especially the radiologists has largely disappeared.

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Experience in such surveys has taught all of us physicians that the public, in becoming more aroused in the interest of their own individual and community health, has turned to their own physicians for more guidance. The program of X-ray chest examinations is now receiving effective expansion and emphasis by the increasing practice of X-ray chest films for all admissions to hospitals. At present over 700 hospitals in the U.S. report that they are X-raying the chests of patients as part of their admission routine. This program can and I believe will be carried to its fullest and most important level when it is brought down to every practicing physician in his office by including in the examination of each new patient an X-ray chest film. In all of our methods and techniques of surveys our objective should be to get the acquired information to the patient and his own physician. At this point the application of the information, the necessary studies indicated and the follow up guidance begins and rests. One of the great weaknesses of the survey is the follow up. Our experience has been similar to that in other communities. Two years after the inception of a community or segmental survey and one year after its completion about one fourth of the positive suspects were still in the process of follow up with pending diagnosis. This is due to a combination of apathy and lack of understanding on the part of the physicians and ignorance, fear or misunderstanding on the part of the patient. By such delay and procrastination any lesion of serious potentialities could, no doubt, progress from one of possible cure to one of hopeless prognosis.

The experience of large community surveys have been that about three per cent of the initial small films have shown such abnormal shadows suggesting significant chest disease, as to indicate further study. In segmental surveys such as schools, industrial groups, military recruits, or hospital admissions the percentage of positive films differs greatly from 1.0 to 6.0 percent depending on age and environmental factors. The most important diseases sought are tuberculosis, cancer and heart conditions, and the percentage of these major pathological findings are influenced greatly by age. The total percentage of reinfection tuberculosis, active and inactive, in general surveys runs from 0.6 percent to 2.5 percent (6 to 25 per 1000) with the active cases averaging about 0.1 percent (1 per 1000). Heart conditions contribute about 1.0 percent. Cancer suspects about 0.1 to 0.2 percent of general survey figures (1 or 2 per 1000) is a major item of importance for detection.

After the survey X-ray chest film the next step includes large diagnostic chest films. Many or most of the persons studied will have no subjective symptoms, or objective physical findings. Therefore, much emphasis must of necessity be placed on the study and interpretation of the X-ray chest films.

In the diagnostic study of X-ray chest films the first point to establish is the localization of the lesion as regards the bones, diaphragms, mediastinum, heart, large vessels, or lungs. When the lesion is visualized in the lung it must be more accurately localized as to hilum area, the lobe or lobes, the particular segment of the lobe involved and its relationship to the pleura. This gross localization with an appreciation of the origin, outline, shape and limitations of the lesion help you to make up your mind whether it is most likely inflammatory or new growth in character.

If the lesion is inflammatory in nature the first question asked is whether it is acute or chronic. Previous X-ray chest films are naturally extremely valuable for comparison in an attempt to decide how long the lesion has been present. The most important question next to tackle is whether the lesion is most likely tuberculous or non-tuberculous. There is no doubt that today many patients are sent to tuberculosis hospitals because an X-ray chest film showed an abnormal density suggestive of tuberculosis.

In critically studying the lesion in the lung the physician should attempt to translate the abnormal densities seen on the X-ray films into terms of pathologic tissue changes. Tuberculous infection with its pathological tissue changes is not static. It is a moving, dynamic process subject to constant change, and it is the progressive change in the lungs that allows of positive roentgenoscopic recognition. In general, the course of tuberculosis is apt to be intermittent with periods of exacerbation and remission, which correspond to recurrent discharges of infection. This leads to multiple lesions in the lungs in different stages of progressive, changing character.

Outline of Pathological changes of Pulmonary Tuberculosis

1. exudate
2. tubercle
3. caseation

absorption	fibrosis	ulceration
	calcification	cavitation

Each lesion produces a corresponding characteristic density on the X-ray film depending upon the histological structure represented whether it is exudate, cavity, caseation, fibrosis or calcification. The quality of the densities on the X-ray films produced by these various tissues and their abnormal changes vary from the least to the heaviest quality as follows: 1. Cavity 2. Normal lung parenchyma 3. Exudate 4. Fibrous scar tissue 5. Caseation 6. Calcification. Thus on the films one may visualize densities of varying quality and by proper interpretation piece them together into the dynamic pathological picture of

pulmonary tuberculosis. The X-ray interpretation presents limitations of presumptive diagnosis when only one density is seen as in an incipient case. Here we see only a single patch of exudate, the etiological diagnosis of which requires more thorough clinical observation and laboratory studies. Likewise a large area of consolidation such as occurs in caseous tuberculous pneumonia, often basal in location, calls for further differential studies. Miliary tuberculosis offers its characteristic picture of small uniform nodules disseminated bilaterally. Let me not seem to claim that all tuberculosis can be diagnosed from the X-ray chest film alone. The X-ray study offers the best and most accurate method of study of lung disease in its location, extent, and character of lesions. Often it is the only evidence detectable in the patient. The application of the knowledge of the pathogenesis of the disease in our study of the films provides a basis for etiological diagnosis. But this diagnosis needs to be verified and amplified by clinical judgment and studies.

The determination of the state of activity of a tuberculous lesion in the lung detected by X-ray examination may at times be very evident and in other instances be very uncertain. The obviously active case is manifested by the X-ray findings of cavity or unmistakable exudate; the clinical signs of toxicity or the laboratory detection of tubercle bacilli in the sputum. At the other end of the scale of activity the obviously inactive case is indicated by the X-ray findings of unmistakable stable lesions showing fibrosis or calcification with no exudate or cavity, the clinical absence of toxic signs and the persistent absence of tubercle bacilli in the sputum. Between these obvious examples there are many cases which must be first classified as activity undetermined. Such cases can only call for conservative handling and cautious care with a sufficient period of clinical observation to allow of studies of the symptoms, repeated X-ray chest films for comparison, and careful laboratory search for tubercle bacilli in sputum or gastric lavage specimens. This may require cultures and animal inoculations of the specimens.

Non-tuberculous lung diseases contribute an appreciable percentage to the abnormal chest films discovered in mass surveys. These can be divided into the chronic infections, chronic granulomatous lesions and new growth. Bronchiectasis is perhaps the most common non-tuberculous disease to need differentiation from tuberculosis. It can exist for a long period of time with few or neglected symptoms and signs. Again it can closely simulate tuberculosis in its clinical manifestations of cough, expectoration, blood spitting and intermittent fever. The differential diagnosis is first suggested by the location and character of the lesion, which is most apt to be located in the bases of the lungs and lingual tip of the upper lobe. The definite diagnosis is further pursued by sputum studies and finally bronchograms and bronchoscopic examinations. Cystic disease should be considered with bronchiectasis because the two conditions so commonly occur together. Cysts may be congenital, the result of maldevelopment of structure, or acquired as the end result of bronchiectasis or other disturbed bronchial function. Cysts can exist to a surprisingly severe degree with few symptoms or signs. The cavities seen on the X-ray chest films are usually thin walled and not accompanied by much infiltration unless infection has been superimposed. Emphysema is also a striking part of the picture as a rule. Lung abscess may occasionally be picked up on a casual survey film, but usually the symptoms are so manifested that the individual seeks medical advice. Fungus infections in varied forms can first be detected on survey films and be most easily confused with tuberculosis. This is especially true with regard to histoplasmosis. In fungus disease while the specific skin tests are valuable, the sputum tests hold the final answer. Lipid pneumonia, usually giving abnormal mottled densities in the bases, but rarely showing a single confluent mass, must be considered especially in young or old individuals who have used long continued oil preparations for nose drops or constipation. All the eliminating laboratory tests must be applied for diagnosis.

Chronic granulomatous diseases of the lungs are exemplified by sarcoidosis, silicosis and beryllium pneumonia. However, it is discouraging, for diagnostic studies, to state that granulomatous lesions, single, or diffuse and multiple as seen on X-ray chest films, can be the result of eighty different pathological conditions. Any one of these diseases can first be detected on mass survey films because the symptoms are slow and insidious in developing. All of these conditions give nodular densities in the lung fields and may show enlargement of the hilum shadows. Sarcoidosis is diagnosed by the absence of tubercle bacilli in the sputum, the common occurrence of negative tuberculin test, the frequent association of lesions in the skin or bones, and finally the biopsy of a lesion in skin or lymph node. Silicosis and beryllium pneumonitis are verified in diagnosis by the industrial history of exposure to the offending substances. The common association of tuberculosis and silicosis is to be recalled. It is also to be remembered that it may be very difficult to determine when tuberculosis complicates silicosis. It may be impossible to establish this diagnosis until tubercle bacilli are found in the sputum.

When a tumor is detected on the initial X-ray chest film the first step in the study of follow up X-ray films is to determine the location of origin of the mass.

Sites of origin can be designated as follows:

1. mediastinum, anterior or posterior
2. hilum
3. lung
4. pleura
5. diaphragm

When the site of origin has been determined the possibilities are reasoned from the type of tissues present in that location. Thus, Anterior Mediastinum Tumors.

1. Lymphoma, lymphosarcoma, Hodgkin's Disease
2. Dermoid cysts and teratomas
3. Fibroma
4. Thymona
5. Thyroid
6. Lipoma
7. Pericardial cyst

Posterior Mediastinal Tumors

1. Neurogenic
2. Bronchiogenic cysts
3. Gastrogenic cysts
4. Enteric cysts

Hilum Tumors

1. Lymphoma, lymphosarcoma, Hodgkin's Disease
2. Bronchogenic carcinoma
3. Bronchogenic cysts

Lung tumors

1. Carcinoma
2. Sarcoma
3. Hamartoma
4. Adenoma
5. Bronchogenic cyst

Pleural Tumors

1. Endothelioma
2. Sarcoma

Diaphragm Tumors

1. Neurogenic
2. Myoma

The exact differential diagnosis of the tumor in any region is only to be made on a thorough clinical study comprehending the pathogenesis of the tumor, its growth, invasive character, limitations, and biopsy of tissue or lymph node when possible.

Some general procedures in the clinical study of the case can be stated with benefit.

Age may be a big factor in determining the probabilities of the kind of infection and especially the type of tumor.

Color. In tuberculosis in the Negro an exudative type of infection is common. In cancer of the lung the ratio in the white is more than two to one that in the colored.

Sex. We know that carcinoma of the lung is three times more frequent in the male than in the female.

Past medical history may give valuable clues, e.g.: pleurisy with effusion, or enlarged lymph nodes (tuberculosis); antecedent malignant tumor (metastases); recent periods of coma or suffocation (aspiration pneumonia), previous injection of opaque oil into bronchi for diagnostic study with residuum in lungs or bronchi (lipoid pneumonia), the presence or previous presence of parasitic disease with lung changes (histoplasmosis). history of asthma or allergic phenomena (Loefflers syndrome).

Family History— especially important to indicate contact in tuberculosis.

Industrial history necessary to establish a diagnosis of silicosis, asbestosis, siderosis, anthracosis, beryllium pneumonitis, or other pneumonoconiosis or gaseous inhalations. Contact with animals including birds, for fungus infections.

Residence. Geographical distribution of certain diseases like histoplasmosis, coccidiodomycosis is established.

Habits. Heavy smoking is receiving more than coincidental or casual relationship to carcinoma. The percentage has been stated to be as high as seven to one.

The tuberculin test can be used in a valuable way to screen possibilities. A positive reaction to 1: 10000 or the 1: 1000 dilution of tuberculin or to P.P.D. affords presumptive evidence in favor tuberculosis, especially in the differentiation of fungus infections. More important a negative reaction to a tuberculin test of low dilution is stronger presumptive evidence that a lesion is not tuberculous.

Sputum examinations are most important and far too often neglected. If no sputum is obtainable with cough, the gastric lavage specimen should be studied with culture or animal inoculation.

Lymph node biopsy when possible is often the positive answer. Search carefully and repeatedly for small node in supraclavicular area or axillae.

Scalene fat biopsy when a nodule cannot be felt, is reportedly useful in some hands and belittled by others.

Bronchograms are imperative for demonstration of bronchiectasis and sometimes useful in showing the position of obstruction to the bronchus.

Bronchoscopic examinations are called for in every case of lung tumor, both for the purpose of tissue biopsy or collection of secretion for cell study. Sometimes bacteriological studies of the gathered secretions give surprising information.

Cell studies of sputum for malignancy is at best positive in only 10 to 15 percent of peripherally placed carcinomas.

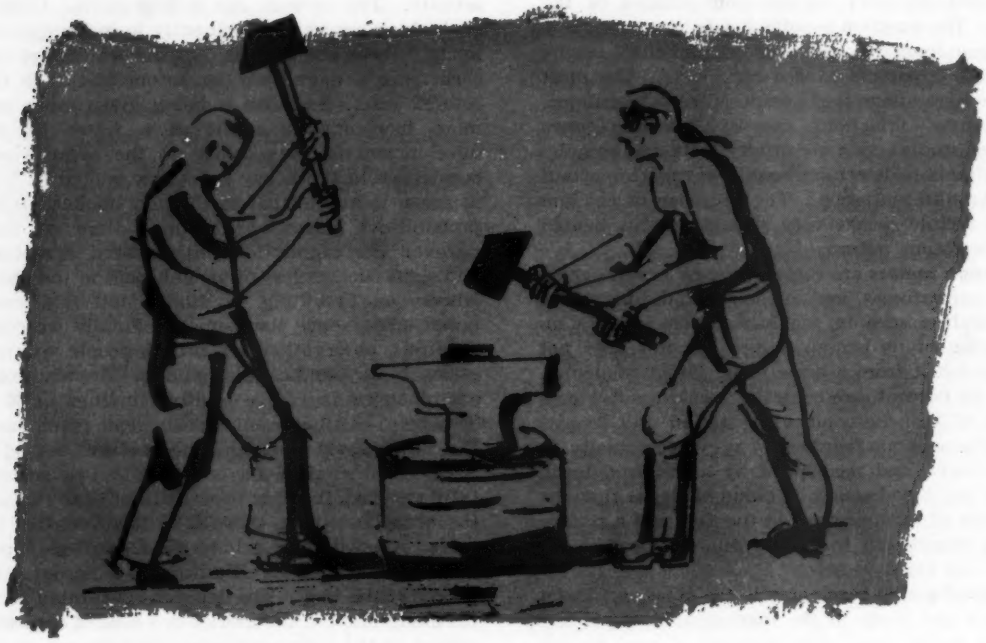
Lung biopsy by intercostal incision is used by some with limited success, but only in diffuse disseminated lesions.

The solitary nodular lesion in the lung represented on the X-ray chest film as the so called "Coin" density calls for special consideration. This

is true because of the extreme difficulty of diagnosis and the very serious potentialities of the lesion. The question to solve is whether the nodule is a granulomatous lesion of inflammatory origin, a benign tumor mass, or a carcinoma. The most common granulomatous nodule is the tuberculoma, but fungus infections can assume this form. Sputum studies, skin sensitivity tests and complement fixation determinations are very important in their differentiation. The presence of calcium in the nodule speaks very strongly for its benign granulomatous nature.

Benign tumors are represented by bronchogenic cysts, hamartomas, and adenoma of the bronchus. Calcification seen in the hamartoma is often an indication of its benign nature but this does not distinguish it from a granuloma. Most adenomas, about 90 percent, are centrally located and within range of bronchoscopic visualization and biopsy. An arteriovenous fistula may very occasionally be encountered and recognized by a localized bruit, a high red blood count, clubbing of finger tips, the presence of telangiectasis in the skin and a pedicle to the density on the X-ray films indicating the supplying blood vessels.

Bronchogenic carcinoma is the chief reason for concern and haste in the management of solitary nodule in the lung. The rapidity of local growth and the stage at which metastases develop may vary very widely. The failure of a small nodule seen on the X-ray to grow appreciably in a year does not exclude carcinoma. Also a small untreated cancer of the lung may develop metastases while the original lesion remains apparently stationary in size. The proportion of the circumscribed "Coin" density seen on the X-ray chest film which represents carcinoma is variously given as 20 to 55 percent. In approaching a diagnosis of such a solitary nodule in the lung we first weigh the probabilities from statistical experiences. Lung cancer is a disease of advancing years. Four fifths of the cases occur between the ages of forty to seventy. The average age is fifty years. Cancer of the lung occurs predominantly in males approximately three out of four. In the white race the occurrence is more than two to one of that in the colored race. Smoking is being given more and more incriminating credit as a factor in the development of lung cancer to the point where occurrence in the heavy smoker is quoted as high as seven to one to that in the non smoker. After probabilities comes possibilities. Here we may uncover unrecognized or disregarded symptoms and signs such as loss of weight, pain in the chest, wheeze on breathing, or find a tell tale small lymph node above the clavicle. Finally we come to direct observations of bronchoscopic examinations, biopsy, and cellular studies. Bronchoscopic examinations may give positive findings in 60 to 70 percent of all cases of bronchogenic carcinomas. But in the early cases, the ones we are seeking in X-ray chest surveys, the ones that can be successfully resected, the lung cancer can be seen through the bronchoscope in only 10 to 15 percent of the cases. A negative bronchoscopic or cytologic report may be of limited significance in ruling out early cancer of the lung. The decision concerning surgical exploration of the thorax in a case of suspected cancer of the lung is of prime importance in our attempt to improve our present day poor survival rate of this disease. Therefore, potentially the most fruitful and promising direction to follow in our efforts is to practice early surgical exploration of the doubtful pulmonary nodule. This advice is sound not only because the mortality rate of such procedures is very small but the lesions which roentgenologically most closely simulate cancer of the lung are usually best managed by surgical excision regardless of their etiology. Between 50 and 75 percent of the solitary nodular lesions of the lung are either granulomas or malignant tumors which should be removed.



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Article

The University and Medical Education

Dr. H. H. Saunderson
President, University of Manitoba

Mr. Chairman, Ladies and Gentlemen:

Had I been able, I would have loved to talk to you about the prognosis and treatment in some 1400 cases of sub-acute arachroid spinneritis. Alas, on this topic, as on so many others better known to medical people, I am delightfully ignorant. Although I am only slightly less ignorant about many of the problems of medical education and research (except that they always seem to cost money to deal with) I felt that I should at least say a little about this important topic. May I at once say to your programme committee that I am profoundly grateful that I was allotted only twenty minutes. This is completely adequate to allow me to say all that I feel qualified to discuss with you; any more generous time allotment would have been an embarrassment to me.

I should start, perhaps, at some of the more practical problems in the field of teaching. One of these which is currently before the Faculty is the advisability of conferring the degree at the end of four years and having the interne year after graduation rather than before. Without prejudging the final decision, I can see a number of cogent practical reasons why this should be done in Manitoba, as is the custom in so many other places. The main difficulty or objection is that the Faculty would lose a measure of the present control over the location where these interneships would be taken. Under the proposed new arrangements some students might be tempted to take interneship at hospitals which offered higher salaries rather than those which offered better training. Frankly, I feel that most students at that stage have too much judgment to imperil the real value of their medical course by electing a present high salary for one year rather than the proper type of interne training. For those few who can't make an intelligent choice, I think that we can rely on the co-operation of the College of Physicians and Surgeons as the licensing authority.

A second practical problem we face is the gradual addition to our staff of full time men in the clinical fields. This change, of course, will involve a substantially greater budget for salaries, which perhaps explains why I spoke of this as a gradual change. There is no thought in my mind that we should ever have a Medical School completely staffed by full time men. Even if we had the money to make that possible, a situation which is completely improbable, I feel that the cause of medical education would not be served by such a step. We have profited greatly, and will continue to profit by using the help of many men in normal practice whose special skills should be used to further the education of the students. But the increasing complexity of medical science and the practical problems inherent in relying almost completely on voluntary help would suggest an increased percentage of full time staff.

Although I have spoken about these new men as full time members of our Faculty, I should, perhaps, say that I am using this term in the way it is normally used in other Medical Schools. Both, those now on our staff and those we hope to add have their special skills and knowledge, and it would seem only proper that these skills and knowledge should be used for the direct relief of the sick as well as for teaching students. When cases are referred to them which lie within their special fields, they should have the time and the opportunity to use their abilities for the well-being of our community.

A third practical problem we have been studying has been the organization of post-graduate training, at least in some fields. There are some who express alarm about the extent of the present trend toward specialization. There is, undoubtedly, an important place in medical practice for men whose training is along more general lines, but we must have a continuing supply of well-trained specialists of many kinds. The Medical Faculty Council has sent forward to the University Senate, and that body has approved, outlines for several post-graduate courses, the completion of which will be marked by the award of a diploma. Facilities for these courses are now generally available here, although the organization and use of these facilities will, undoubtedly, raise some problems before the first group of students completes its work.

In thinking about medical education, it is obviously not good enough to speak only of the practical problems we are currently studying. We must think also of the broader educational philosophy which will give us some guidance in our detailed decisions. We must think of the way we should be going.

A person reading a university calendar would note in it a prescription of courses in the several departments, running in our case the alphabetical gamut from anatomy to surgery. Each of these departments has its own disciplines and sub-disciplines, and each makes its own contribution to medical education. But I feel that the medical course must be something more than a measured portion of anatomy and physiology, pathology and

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obstetrics, psychiatry and otorhino-laryngology and the other departmental subjects. These must be integrated into a unified approach to the welfare of man. We must place our emphasis on the patient, not on the subject. There is a discernible trend toward thinking of these several subjects not as subjects in themselves but as areas of light which can shed their several illuminations on the structure and functioning of the living individual.

This trend toward a fusing of the light from these disciplines is resulting in a gradual shift in our course program. There is no longer the sharp split between pre-clinical and clinical work; students are having the opportunity to make direct contact with hospital patients earlier in their course. This change will, I think, make the several disciplines more real to the student, and enable the departments to make a more direct contribution to his education.

Although teaching is one of our main functions, the University should also be actively interested in research. We are. The change is perhaps less obvious to those who have been here while it occurred than to me, who was away for several years. On my return, I felt a new enthusiasm and a new order of activity and productivity. It was only when I started to prepare this talk that I began to gather any numerical data to support my belief. In the academic year 1945-46, there were published by the medical faculty some 25 research papers or books. Last year the number was over 100. Grants in aid of research from outside sources have shown an even greater growth. In the session 1945-46, I find record of grants from outside sources amounting to less than $2500.00 for medical projects. Last year, the corresponding figure was over $100,000.00. The figures for this year will be even higher, the present figures running at above $150,000.00. Support of this order from organizations outside the University indicates that these people realize the high quality of the work being done here, and are prepared to support it on this generous scale.

We in the University are naturally very pleased at this help, and we hope that it continues on an even larger scale. In speaking to this group, which is so deeply interested in the welfare of the University, it would be less than realistic if I didn't mention one problem, which this outside generosity poses for us.

Those of you who have had experience with research realize that the direct costs of research (which are paid for by these grants) are always accompanied by indirect costs. Few grants make any provision for these indirect costs, so they must be met out of the general University budget. That budget is always strained as we try to improve the present very low salaries and provide on a slighly less parsimonious scale for general operating supplies. Our staff is a very strong one, and if we are to keep them, and to add to their numbers, we must obtain a larger income for general budget purposes. Much of this additional income will necessarily come from governmental sources and from student fees, but I am sure that we should be receiving from our graduates and other friends of the University a substantially larger amount than they are now contributing to the general support of the University. Our graduates particularly should recognize more tangibly the contribution which others have made in meeting the costs of their education, and pass on to those coming later some of the help which they received. Many students feel that the fees they pay (which are high compared to their ability to pay them) cover the costs of their education. This is not so. Student fees in the University pay only 30% of the costs, the other 70% being met by other sources of income. It is not unreasonable to suggest that some small portion of the increased earnings made possible by a University education should be returned by those who benefit directly from that education, to enable the University to carry on the work it is trying to do. You will no doubt hear more about this from the Alumni Association this winter.

In addition to money, research needs space. I am happy to say that the new Medical Building extension is almost completed, and this should make available not only increased and improved teaching space, but also a substantial increase in the amount of room available for research laboratories. Some of this will be in the new building, but additional research laboratories are being provided in the old building in space vacated by departments moving into the new building.

With new or rebuilt laboratories and with additional equipment which can now be accommodated, I am looking forward to an even greater development of our research activities which mean so much to the staff and to the medical community at large.

You will have noticed that my remarks on the Medical School, both on the teaching side and on the research side, have been optimistic. This optimism is justified. In spite of the difficulties we all know about, we have an excellent School. The staff, both full-time and part-time, are making a very valuable contribution in time and talent to the cause of better medical education. This contribution they are making often at great personal sacrifice. Their record of service in the past has been a memorable one, and I have no doubt that the future will hold even greater promise for medical education and research in Manitoba.

Medical History

Medical Place-Names in Manitoba

Ross Mitchell

The November 1955 number of the always interesting Quarterly Historical Bulletin of the Calgary Associate Clinic had an article on medical place-names in Alberta. Provincial pride led to examination of "Place-Names of Manitoba", published in 1933 by the Geographic Board of Canada. The provincial representative of the Board was Mr. W. J. Healy, then Provincial Librarian. The late Dr. D. A. Stewart was also actively interested and contributed information embodied in the volume which amply repays study.

The place-names reflect the history of the province and the various ethnic groups which form its population. Indian names such as Minnedosa, Manitoba, Wawanesa, Pinawa, Assiniboine are descriptive and mellifluous. Then there are French names; e.g. Letellier, Notre-Dame de Lourdes, Giroux, Lazare; Scottish, e.g., Strathclair, Gretna, Kildonan; Mennonite such as Kleefeld, Rosenburg, Steinbach; Icelandic, such as Baldur, Hnausa, Husavik; Irish, e.g., Killarney, Clandeboye; English, e.g., Somerset, Headingly, Fairford, Dynevor; Ukrainian and Russian such as Narol, Olga and Tolstoi.

The medical place-names are:

Bird's Hill, after the Hon. Curtis James Bird, M.D. (1838-1876), speaker of the first legislative Assembly. Bird's Hill was part of the estate of James Curtis Bird, Chief Factor, Hudson's Bay Company and father of Dr. Curtis Bird.

Bunn Lake, after John Bunn, M.D. (died 1861) first native-born doctor of the Red River, Sheriff of Assiniboia.

Harrison municipality commemorates Hon. D. H. Harrison, M.D., the only medical premier of Manitoba (1887).

Orok, a station on the Hudson Bay Railway is named after Dr. R. D. Orok, M.L.A., who practised at The Pas.

Schaffner station on the C.P.R., gets its name from Hon. F. L. Schaffner, M.D., M.P., who practised at Boissevain.

Crandall village is named after Dr. J. B. Crandall who practised in that area.

Bissett P.O. was named after Dr. E. D. R. Bissett, M.P. for Springfield constituency 1926-30.

Baragar, a C.P.R. station, gets its name from Dr. C. A. Baragar, Medical Superintendent, Brandon Mental Hospital.

Colert Lake, the artificial body of water formed by damming Cheval creek, is named for Dr. W. M. Colert of Morden.

Schultz Street in Winnipeg commemorates Sir John Christian Schultz, M.D., Lieutenant-Governor of Manitoba, 1888-1896, who owned property in north Winnipeg.

Clearwater Sanatorium is the name of a post office in Churchill constituency.

Perhaps our readers can add other medical place-names.

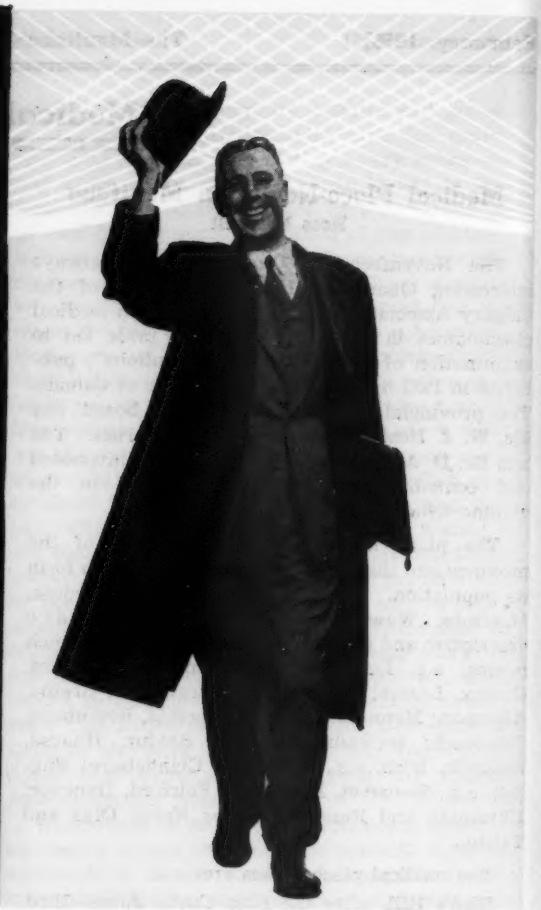
Obituary

Dr. Wilfrid S. Peters

Dr. Wilfrid S. Peters died at Brandon on January 8. Born at Spencerville, Ontario, 67 years ago, he came west with his family in 1890, graduated from Manitoba Medical College in 1910 and practised at Souris and Brandon. He was a Fellow of the American College of Surgeons and President of the Manitoba Medical Association in 1938-39. In his later years he restricted his practice to obstetrics and gynecology.

Dr. Thomas Turnbull

Dr. Thomas Turnbull, a resident of Winnipeg for over 50 years and one of the first eye specialists in the city, died on January 11 at the age of 83. Born at Stratford, Ontario, he graduated in medicine from McGill University in 1899. For many years he was associated in practice with the late Dr. J. W. Good. He was a Fellow of the American College of Surgeons. He is survived by his wife.



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Editorial

S. Vaisrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

Fate of Iron

The case reports by Bennett, Vaisrub, and Karlinsky, of two uncommon disorders of iron metabolism, published in this issue, can hardly fail to arouse interest and provoke thought. Both, primary and secondary hemochromatosis are relatively rare. In fact, but a few years ago they were of the stuff that "news" is made of. Long descriptive titles, as they appear in these reports, emphasizing some unusual feature to justify their reportability, would have then been superfluous. No "spicing" to enhance their palatability would have been thought necessary. (Would a barker in a side show pause to enlarge upon an unusual curl in the goatee of the bearded lady?) In recent years, however, a considerable number of papers dealing with the subject have appeared in print, thus detracting somewhat from their novelty. It is, likely, this consideration that made the reporters of these cases look for "angles". Luckily, the latter were easily found, for these cases present some very unusual features. It would, however, be erroneous to assume that their value is predicated upon novelty or nonconformity. They are valuable if for no other reason than that they make us pause, reflect and "brush up" on some problems of the metabolism of iron, particularly those concerned with iron overload.

The metabolism of iron is unique in that it occurs in a closed system, both absorption and excretion being minimal. Less than 10% of the ingested iron is absorbed and even less than that excreted in 24 hours. Out of the 27 mgms. of iron that the body utilizes per day, 20 are supplied by the destroyed red blood cells, and the remainder by storage iron, and the iron that is newly absorbed.

The absorption of iron occurs in the intestinal wall. It is mediated by the concentration of ferritin, an iron-containing protein in the intestinal mucosa, derived from apoferritin, a protein which contains no iron. When the level of ferrous iron and the concentration of ferritin in the mucosal cells rise, as they do after ingestion of iron, no further absorption takes place for 12 — 24 hours. Conversely in iron deficiency anemia, where the concentration of ferritin is low, absorption may be increased tenfold. The "mucosal block", thus, acts as a delicate homeostatic mechanism, adjusting absorption according to need.

The above outline, of course, is an oversimplification. There are many other factors that enter into the picture. The efficiency of the breakdown of ingested ferric hydroxide and ferric organic chelates into free ferric ions or loose chelates by the hydrochloric acid of the gastric juice and the organic acids of food stuffs, the subsequent conversion of ferric iron to a more soluble and absorbable ferrous form by reducing substances (ascorbic acid, cysteine), the phosphate content of the food, the state of the bowel wall, the oxygen tension, and other less well known factors exercise a modifying effect.

The absorbed ferrous iron leaving the intestine forms, after prompt autooxidation, a complex with transferring (siderophilin), an iron-binding beta-globulin, and is subsequently stored in the body in the form of ferritin and hemosiderin, the latter containing 35% of iron combined with protein. The level of the iron in the serum and its storage in the tissues are in a state of dynamic equilibrium, being mutually interdependent as well as dependent upon absorption from the gastro-intestinal tract, hemoglobin breakdown, uptake by bone marrow and availability of transferrin. The normal range of serum iron is between 50 and 150 micrograms per 100 cc. This amount binds only about one third of the usually available transferrin, thus leaving about 150 - 250 micrograms in an unsaturated state—latent iron-binding capacity, (L.I.B.C.). The ratio of the unsaturated transferrin (L.I.B.C.) to the total iron-binding capacity (T.I.B.C.) is often used as a convenient index of iron turnover. Methods used for the study of the latter, chemical balance studies as well as test procedures with radioiron, have helped considerably toward understanding some of the problems of normal as well as deranged metabolism of iron.

The application of these methods to the problem of idiopathic hemochromatosis has shed some light on its etiology. Patterson and Ettinger (Am. J. Med. 15, 518, 1953) have found that, while normal subjects absorbed only 1.5 - 6% of ingested radioiron, patients with idiopathic hemochromatosis absorbed 20 - 45%. These figures are in agreement with the results of balance studies by chemical methods and tend to support the view that the basis of the disorder is a congenital defect in the absorptive mechanism, a breakdown of the "mucosal block". Whether an additional factor of tissue avidity for iron is also operative, is a moot point.

Unlike primary hemochromatosis, the secondary, exogenous one, particularly the transfusional variety, presents no problem with regard to etiology. Its pathology and pathophysiology, however, are still left with many unanswered questions. Some of them, perhaps, the most important ones relate to the nature of the difference between the primary and secondary types. Is the difference

fundamental, or is it merely one of duration? Are the diabetis, cirrhosis, and hypogonadism of the primary variety due to a selective deposition of iron in the involved organs in contradistinction to the secondary one, where symptoms are absent because these organs are spared? Or is the distribution of iron similar in both conditions, the pathological and clinical differences being due to differences in the duration of the process of accumulation of iron, which is much longer in primary hemochromatosis, beginning as it does in early life? The orthodox view until recently favored the first explanation. Kleckner and his associates (Am. J. of Med. 16, 383, 1954) reaffirm this concept and reinforce it with impressive evidence. On the other hand Morningstar (A. M. A. Arch. of Path. 59, 355, 1955) is equally emphatic in his assertion that severe exogenous hemochromatosis is indistinguishable from the idiopathic variety. He concludes from his case that the degree of pigmentation and fibrosis is a function of the number of transfusions. (One of his cases, who received 254 blood transfusions, manifested all the features of primary hemochromatosis cirrhosis, diabetis and hypogonadism.)

It becomes, thus, apparent that until an iron-clad theory emerges to resolve the above perplexities, every new case report will be welcome as a potential clue to some of the mysteries of iron overload. The more clues, the sooner the solution. "Where there is a will there is a way," especially if the will is the "will of iron".

Ed.

Manitoba's Medical Men
XXIII. Annual Meeting, 1955

The annual meeting of the Manitoba Medical Association was held in October and the attendance was large.

A letter was sent to the Minister of Health and Public Welfare indicating that the Association was favorably impressed with the Salk Vaccine program as carried out by the Department of Health in this province.

The chairman of the Committee on Economics gave his report. The last item dealt with a motion from the General Practitioners' Association that they were in favor of a change in the fee schedule of the Manitoba Medical Service. They made a motion that there be one fee across the boards, the Manitoba Medical Association schedule of fees, with specialists having the privilege of extra billing. The thinking behind this motion was that one premium entitles a subscriber to one type of service. This principle is inherent in other prepaid medical plans. Had this passed the administration would have been simplified, the block system would have been eliminated and specialists could have charged patients extra for services rendered. It would also have eliminated any basis for the assertion that the general practitioner was subsidizing the specialist and the poor patient subsidizing the wealthy patient.

Prior to the meeting of the Manitoba Medical Association some of the specialist groups were opposed to the motion. The Obstetrics and Gynecological Section of the Winnipeg Medical Society passed a motion: "That the present system of differential tariffs in the Manitoba Medical Service be retained". The Paediatric group agreed that if these differential tariffs are abolished it would support a motion to the effect: "That the Manitoba Medical Service should be a service for General Practitioners only and that all specialist services be withdrawn". The Surgical Section passed the following resolution: "The Surgical Section of the Manitoba Medical Association is in complete sympathy with the resolution of the General Practitioners' Association as a protest in relation to Manitoba Medical Service fees. This matter, however, should be referred to a study committee on fees for a thorough investigation and study, and the recommendations of this committee should be considered before any action is taken in this connection".

Since there seemed to be some danger of splitting the medical profession into two groups, an amendment to the motion was moved and carried without much discussion: The whole content of the amendment to the motion is as follows:

"WHEREAS The submission of the general practitioners to the Manitoba Medical Association at the Annual Meeting in 1951 was never implemented.

"All other prepaid medical care plans on this continent follow the principle of one fee for one service.

"The effect on general practice has been considerable on account of heavy enrolment of subscribers to M.M.S. in Winnipeg.

"The M.M.S. is recognized as the economic arm of most sections of the medical profession in Manitoba.

"The extension of M.M.S. into rural Manitoba is increasing.

"The general practitioners of Manitoba have 487 doctors on the M.M.S. roster out of 746.

"There has not been any general revision of the M.M.S. fee tariff since 1949.

"The economic future of all physicians will be determined by Government decisions as to the price that Government is prepared to pay for medical care.

"The principles on which a fee tariff are based need periodic re-examination for re-statement, modification or elimination in the light of principles found acceptable elsewhere in Canada.

"Other sections of the M.M.S. medical Membership may have views, not yet ascertained, as to the adequacy of the current fee tariff.

"NOW THEREFORE BE IT RESOLVED that this Annual Meeting request that the Executive of the Manitoba Medical Association set up a special Commission consisting of three members appointed by the Executive of the G.P.A.M. and three members from the specialist blocs of the M.M.S. appointed by the President of the M.M.A. after consultation with the specialist blocs; and the chairman of this Commission be appointed by the Executive Committee of the Manitoba Medical Association in consultation with and the approval of the members of this Commission."

The Terms of Reference of this Commission shall be to:

1. Elucidate the principles on which the current M.M.S. fee tariff is based;

2. Examine the principles behind the fee tariffs on prepaid medical care plans in Canada and the U.S.A.;

3. Examine the feasibility of a ceiling limit on incomes of subscribers and right of extra billing by all medical members of the M.M.S. to subscribers with incomes in excess of an amount to be determined by M.M.S. after statistical studies and all other such relevant matters;

4. Examine the principles on which negotiation with Government, acceptable to the medical profession shall be based.

5. Report on the above and matters relevant to it to the Executive of the M.M.A. by a date not later than April 1, 1956, and through them to each individual member of the M.M.A. with approval of any recommendations by vote, either at a special general meeting of the M.M.A., mailed ballot or at the next meeting of this Association.

6. Present the proposals of this Commission, when so approved to the M.M.S. with recommendations for their implementation.

Report of the Chairman of the M.M.S.

Literally at the eleventh hour the chairman of the board of trustees of the Manitoba Medical Service (an autonomous body with an annual income of about $4,500,000.00) was invited to give his report. The report was very comprehensive and indicated that the board of trustees of the Manitoba Medical Service had devoted a great deal of time and effort on behalf of the members of the profession. Since about 50 to 60% of the doctors income is derived from M.M.S. fees, the doctors are vitally interested in what the board has done and what they propose to do.

The chairman of the board presented on behalf of the board of trustees, six specific recommendations. The first five recommendations were approved by the Manitoba Medical Association.

The sixth recommendation was the institution of the principle of "co-insurance" in all Manitoba Medical Service contracts. The substance of this was as follows:

"That there be introduced into all contracts of coverage the concept of "service charges", often called "deductibles" or co-operative payments.

"This charge should not be levied for in-hospital care or the first office or hour call per person in any one month. The amount of the charge is a matter of policy, but a trial could be made of $1.00 for each item of service in the General Practitioner's column, and $2.00 for each item of service in the specialist column and the Manitoba Medical Service Tariff where the fees are identical, the deductible could be one dollar".

The profession at large was quite unprepared to understand this involved clause or to assess the implications that it would have in medical practice. Stripped of all verbiage such as "service charges" "deductibles" and other complex insurance terms, the clause was intended to give doctors the privilege of extra-billing for second and subsequent house calls, and second and subsequent office calls, but no extra-billing would be permitted for hospital cases. Further, the extra billing would be limited to $2.00 for the specialist and $1.00 for the general practitioner. The many possible effects that might have happened had this board recommendation passed, are as follows:

1. About $614,000.00 extra might have been collected by the doctors.

2. Many patients might have been deterred from seeing the doctor a second time.

3. Two sets of bills would have to be issued for one service.

4. Hospital admissions would have been demanded by patients to avoid extra billings by the doctor.

Although no action was taken on this clause the board of trustees of the M.M.S. can still write this into their contracts if they consider this to be in the best interests of the subscribers and doctors, keeping in mind that the M.M.S. was started to give full coverage to people in average income groups.

A motion was made and carried that the Manitoba Medical Service prepare their annual report one month ahead of the annual meeting of the Manitoba Medical Association in order to give the members of the Association an opportunity of studying the problems of the M.M.S.

One year ago the Manitoba Medical Association went on record as endorsing the principle of experience rating for groups underwritten by the Manitoba Medical Service, where effective and practicable. It was not clear from the report what specific measures had been taken in this regard and what the effect had been during the last year.

An excellent suggestion by the board is being implemented to the effect "that in future all subscribers will receive monthly statements of moneys paid to their doctors for service rendered, instead

of a total annual statement for income tax purposes.

In his closing remarks the chairman of the board asked for the whole-hearted support and loyalty of the profession to the Manitoba Medical Service and indicated that his duties as chairman and the duties of the executive director were to co-ordinate and execute the wishes of the board of trustees of the Manitoba Medical Service.

L. A. Sigurdson, M.D.

Medical Library Accessions

A list of books received by the Medical Library, July, 1954 to June, 1955 was distributed in December to all hospitals and clinics in Manitoba, with the request that it be circulated or posted up. This list is issued annually or semi-annually; one covering the Library's accessions from July to December, 1955 will be sent out shortly.

Clinical Reviews

Mayo Clinic and Mayo Foundation
April 9, 10 and 11, 1956, Rochester, Minn.

A 3-day program entitled "Clinical Reviews" will be presented April 9, 10 and 11, 1956, at Rochester, Minnesota. The presentations will be given by staff members of the Mayo Clinic and the Mayo Foundation for Medical Education and Research. The meeting will be devoted to lectures and discussions on problems of current interest in general medicine and surgery.

The number of physicians who can be accommodated is necessarily limited. Those wishing to attend should communicate with Mr. R. C. Roesler, Mayo Clinic, Rochester, Minnesota. There are no fees of any kind.

General Medical Council of Great Britain . . . . .

Advice has been received from the General Medical Council of the United Kingdom that graduates of the University of Manitoba who qualified after October 20th, 1948, may properly be regarded as having had satisfactory experience in a resident medical capacity to entitle them to full registration by reciprocity, if they are fully registered in the Province of Manitoba.

Graduates of the University of Manitoba who qualified before that date, or persons other than graduates of the University of Manitoba who apply for full registration in the United Kingdom by virtue of membership in the College of Physicians and Surgeons of Manitoba, will be asked to produce evidence of actual hospital experience.

The College of Physicians and Surgeons of Manitoba,
M. T. Macfarland, Registrar.

Book Reviews

Surgery of the Ambulatory Patient, L. K. Ferguson, M.D., F.A.C.S., Professor of Surgery, Graduate School of the University of Pennsylvania. J. B. Lippincott Company, Montreal. 3rd Edition. 894 pages. 6664 illus. $12.00.

This concise textbook has been completely revised by Dr. Ferguson and his associates. The original purpose of the book has been maintained: namely to provide a text for the guidance of physicians called upon to treat surgical lesions in ambulatory patients. It is especially useful for general practitioners in treating patients who go to an office or a casualty department.

The text has a new double-column format for increased ease in reading. It is divided into three sections; Surgical Principles and Lesions, Regional Surgery, and the Musculoskeletal System.

The chapters on Anesthesia and Urology have been revised by new authors. Dr. E. J. Pulaski has provided a good evaluation of the antibiotics and their uses.

A few of the many important additions include: the use of the adrenal hormone preparations in bursitis, trigger finger, etc; new concepts in the management of thermal injuries; and the management of shock after trauma. Many new illustrations have been added. Dr. Louis Kaplan's section on fractures is excellent.

The text is highly recommended.

J. M.

Handbook of Pediatrics, by Henry K. Silver, C. Henry Kempe, and Henry B. Bruyn.

In this most concise and amazingly complete pocket-size textbook the authors (three "Henrys"), who are well recognized pediatricians from Yale and California, have done exactly what they proposed in the one page preface: " . . . endeavored to present to the practicing physician and medical student a concise and readily available digest of the material necessary for the diagnosis and management of pediatric disorders".

Not a square inch of space is wasted nor an extra word used in the book. It is as complete in its abridged form as any of the standard pediatric textbooks, and surprisingly current with such discussions as the dangers of oxygen in retrolental fibroplasia. The tables are useful, and the description of procedures and tests is very clear in spite of its brevity. The tiny print requires sharp eyes for reading. This up-to-date glove-compartment pediatric handbook will be found useful by the practitioner and interne alike.

F. J. C.

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Social News

Reported by K. Borthwick-Leslie, M.D.

'Tis a time for congratulations . . . Individual Medical Members have been recognized in many fields.

The special convocation ceremony held January 20th was colorful and impressive, with the conferring of Doctor of Laws (honoris causa) on our own well beloved Drs. Adam Menzies, M.C. War Cross (Czechoslovakia), M.D., C.M. of Morden, Man., and Rosslyn B. Mitchell, B.A., M.D., C.M., F.A.C.S., F.R.C.P. (C) and Dr. G. Donald Cameron, M.D., C.M., D.P.H., LL.D. (Queen's), Deputy Minister of National Health, Ottawa.

The official opening of the new wing of the Medical College, Auditorium and Library, Canteen, etc. in conjunction with the Convocation was interesting and certainly a wonderful demonstration of the end results of Dean Bell and his co-workers' persistent labors. The building is beautiful. Particularly attractive is the Doctors' Reading Room in the library, furnished by the Winnipeg Medical Society.

♦

Sincere congratulations also to our Tom "Citation" Casey, definitely the most popular choice of "Citizen of the Year".

♦

Dr. Arthur E. Childs, has been elected a Fellow of the American College of Radiology in recognition of his contribution to Medicine and radiology. The investiture will be made February 10th in Chicago at the college's annual meeting.

♦

I just cannot let pass those "Leap Year" members pictured in the Tribune. Apparently it's open season for Dr. John Hildes and Louis Cherniak (your cat is particularly photogenic, Louis), but how come Buzz Bell didn't appear with all the other handsome eligibles? OK girls, no holds barred.

Thank you Dr. Hodgson, for your good wishes and friendly note, announcing the departure from Steinbach to Pincher Creek, Alta. of Dr. and Mrs. Murray Hodgson, Brian and Marion, with the extra notice of the arrival of Margaret Ann, December 18th, at Pincher Creek.

Dr. Hodgson is now associated with Dr. Lorne Collins. Everyone healthy and happy.

♦

Welcome to Dr. and Mrs. Meikle and children who arrived from Edinburgh December 6th. Dr. Meikle is associated with St. Boniface Hospital.

♦

Rumor has it that there will be a mass exodus of the "Orthopods" to the Annual meeting in Chicago the end of January. Understand that Drs. Ryan, Deacon, Tucker, Hollenberg and several others are taking off, both to be scientifically minded and escape Manitoba winter scenery for awhile.

♦

Dr. and Mrs. Lyle Henry are happy to announce the arrival of Ellen Joy, December 29th. Sister for Gail and Bobby.

♦

Dr. and Mrs. John L. Davies, on December 22, 1955 welcomed their daughter Margaret Glenys.

♦

Dr. and Mrs. P. J. Miller, Humboldt, Sask., announce the birth of Patrick Richard.

♦

Gleaned from Nurse's notes:

(1) Man with renal colic is reported as "rollicking" in bed with pain at 5 a.m.

(2) French nurse on duty with a seriously ill priest, reports "caught" Father ..... feeling "fonney" two times last night.

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DOSAGE: One tablet before retiring

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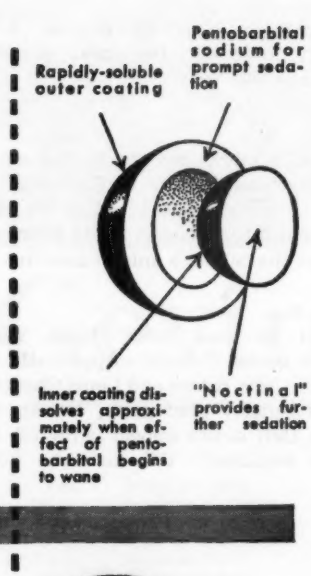
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"Noctinal" provides further sedation

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Association Page

Reported by M. T. Macfarland, M.D.

Income Tax Information

Individuals whose income—(a) is derived from carrying on a business or profession (other than farming); (b) is derived from investments; or (c) is more than 25% derived from sources other than salary or wages, are required to pay their estimated tax by quarterly instalments during such year. Each payment must be sent in with Instalment Remittance Form T.7-B Individuals. Any balance of tax is payable with interest with the T.-1 General return which is due to be filed on or before April 30 of the succeeding year.

The following timetable indicates the returns required.

A. Doctors **Not** receiving salaries amounting to ¾ of income:

Date Due	Forms to be Used
March 31	T.7-B Individuals
April 30	T.1 General
	(Note: Only doctors deriving their full professional income from salaries may use Form T.1 Short).
June 30	T.7-B Individuals
September 30	T.7-B Individuals
December 31	T.7-B Individuals

B. Doctors receiving salaries amounting to ¾ or more of income:

Date Due	Forms to be Used
April 30	T.1 General
	(Note: Doctors deriving their full professional income from salaries may use Form T.1 Short).

Whenever Status is changed* T.D.-1.

*With respect to new employer, marital status, dependents.

Doctors who pay salaries to their own employees are required to send in Form T.-4 by the end of February each year.

DOMINION INCOME TAX RETURNS BY MEMBERS OF THE MEDICAL PROFESSION

As a matter of guidance to the medical profession and to bring about a greater uniformity in the data to be furnished to the Taxation Division of the Department of National Revenue in the annual Income Tax Returns to be filed, the following matters are set out:

Income

1. There should be maintained by the doctor an accurate record of income received (in dated cash or day book), both as fees from his profession and by way of investment income. The record should be clear and capable of being readily checked against the return filed. It may be maintained on cards or in books kept for the purpose.

Expenses

2. Under the heading of expenses the following accounts should be maintained and records supported by vouchers kept available for checking purposes:

(a) Medical, surgical and like supplies;

(b) Office help, nurse, maid and bookkeeper; laundry and malpractice insurance premiums.

**(If in a private home and the maid would clean the office, a portion of her wages could be counted as an expense — yearly maximum $120.00.)*

(It is to be noted that the Income Tax Act does not allow as a deduction a salary paid by a husband to a wife or vice versa. Such amount, if paid, is to be added back to the income).

(c) Telephone expenses;

(d) Assistants' fees: The names and addresses of the assistants to whom fees are paid should be furnished. This information is to be given each year on Income Tax form known as Form T.4, obtainable from your District Income Tax Office;

(e) Rentals paid: The name and address of the owner (preferably) or agent of the rental premises should be furnished (see (i));

(f) Postage and stationery:

(g) Depreciation: A description of the treatment of depreciation may be found on page four of the Income Tax Return form T.1 General under the Part XI Method.

The method of computing depreciation for tax purposes is the same as that used last year and you should have no difficulty if you have a copy of last year's return available.

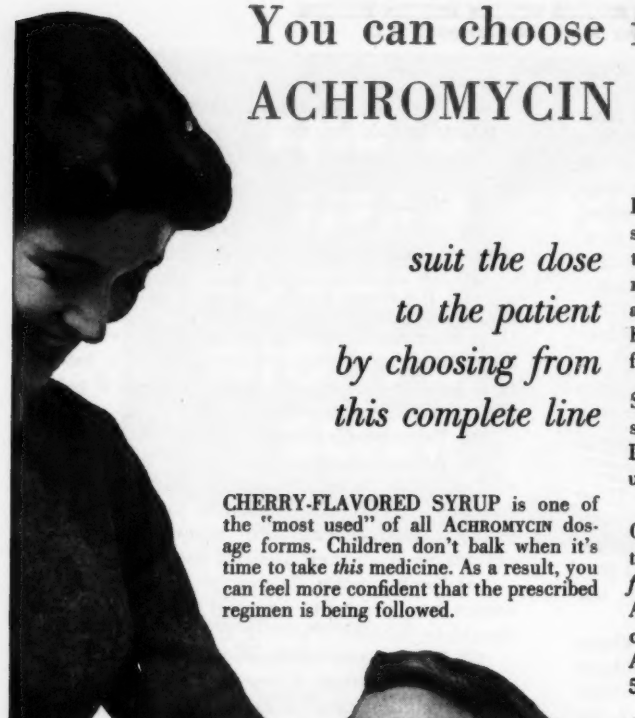
Simply carry forward the balance remaining in each class after deducting last year's allowance. Add to this figure the cost of any new equipment purchased and deduct the proceeds from any disposal of property in each class. The rate you wish to use not exceeding the maximum rate (see below) is applied to this new balance for each class to obtain the depreciation you may claim this year.

The schedule on page four of the Income Tax Return is reproduced below for your information. Column (6) does not apply to doctors, the other columns are self-explanatory.

The maximum rates for the classes of equipment most used by doctors follow:

Capital Item	Class	Annual Maximum Depreciation
Medical Equipment:		
(a) Instruments Costing over $50 Each and Medical Apparatus of Every Type	8	20%
(b) Instruments Under $50 Each	12	100%
Office Furniture and Equipment	8	20%
Motor Car	10	30%

You can choose from more than 20 ACHROMYCIN HCl dosage forms



suit the dose to the patient by choosing from this complete line

From capsules to ointment to syrup, there's a special ACHROMYCIN* Tetracycline dosage form to suit virtually every medical requirement. No matter which one you employ, your patient gets all the benefits of this effective antibiotic: true broad-spectrum activity, prompt control of infection, negligible side effects.

Specify ACHROMYCIN! Every gram of this outstanding drug is made under rigid controls in Lederle's *own* laboratories, and offered only under the Lederle label.

CHERRY-FLAVORED SYRUP is one of the "most used" of all ACHROMYCIN dosage forms. Children don't balk when it's time to take *this* medicine. As a result, you can feel more confident that the prescribed regimen is being followed.

ONLY LEDERLE offers tetracycline in a *dry-filled* sealed capsule. Advantage: rapid and complete absorption. Available in potencies of 50, 100, and 250 mg.

TO INITIATE THERAPY, many physicians use ACHROMYCIN Intramuscular. The vial of 100 mg. is as convenient to use in the home as in the office.

TOPICAL INFECTIONS often respond dramatically to treatment with ACHROMYCIN Ointment 3%. This bland-base cream is offered in ½ and 1 oz. tubes.

The Lederle Representative or your local pharmacist will gladly tell you about the many other ACHROMYCIN dosage forms.

LEDERLE LABORATORIES DIVISION
NORTH AMERICAN Cyanamid LIMITED
MONTREAL, QUEBEC

Lederle

*REG. TRADE-MARK

Building (Residence Used Both as Dwelling and Office) 3 5%

**(If the building is frame or stucco on frame, it could be classified under No. 6 at 10% depreciation.)*

Instruments costing less than $50.00 each belong in class 12 and have a maximum allowance rate of 100%. They should not be included in expenses but should be recorded as additions in columns 3 of the schedule.

Where a doctor practises from a house which he owns and resides in, the allowance may be claimed as above on a portion of the cost of the residence, excluding land. For example if the residence were a brick building costing $12,000 and one-third of the space were used for the office, the doctor would use $4,000 as the business portion of the cost and apply the building rate of 5% to determine the maximum depreciation allowable in the first year.

For further information on the subject you may refer to the Regulations or you may consult your District Income Tax Office.

The doctor should be prepared to demonstrate, if called upon to do so, that his apportionment of any particular item is in accordance with the facts relative to that item.

The above allowances will not exceed one-third of the total house expenses or rental unless it can be shown that a greater allowance should be made for professional purposes.

(j) Sundry expenses (not otherwise classified) —The expenses charged to this account should be capable of analyses and supported by records.

Claims for donations paid to charitable organizations will be allowed up to 10% of the net income upon submission of receipts to your Income Tax Office. This is provided for in the Act.

The annual dues paid to governing bodies under which authority to practise is issued and membership association fees, to be recorded on the return, will be admitted as a charge. Initiation fees and the cost of attending post-graduate courses will not be allowed.

(1) Class Number	(2) Undepreciated Capital Cost at Beginning of 1951 (Col. 10 of 1950 return)	(3) Cost of Additions During 1951	(4) Proceeds from Disposals During 1951	(5) Undepreciated Capital Cost before 1951 Allowance (Col. 2 plus 3, less 4)	(6) Net Deferred Assets	(7) Amount on which 1951 Allowance is Calculated (Col. 5 less Col. 6)	(8) Rate %	(9) Capital Cost Allowance for 1951	(10) Undepreciated Capital Cost Less Deferred Assets (Col. 7 less Col. 9)

(h) Automobile expenses: (One Car). This account will include cost of license, oil, gasoline, grease, insurance, garage charges and repairs;

The capital cost allowance is restricted to the car used in professional practice and does not apply to cars for personal use.

Only that portion of the total automobile expense incurred in earning the income from the practice may be claimed as an expense and therefore the total expense must be reduced by the portion applicable to your personal use.

The mileage rate permitted in years prior to 1950 may no longer be used to estimate the automobile expenses.

(i) Proportional expenses of doctors practising from their residence:

(a) Owned by the doctor. Where a doctor practises from a house which he owns and as well resides in, a proportionate allowance of house expenses will be given for the study, laboratory, office and waiting room space, on the basis that this space bears to the total space of the residence. The charges cover taxes, light, heat, insurance, repairs, capital cost allowance, and interest on mortgage (name and address of mortgagee to be stated);

(b) Rented by the doctor. Only the rent and other expenses borne by the doctor such as heat and light will be apportioned inasmuch as the owner takes care of other expenses.

(k) Carrying charges: It is now possible under Income Tax Act, Section 13, that if an investment loss is incurred, it is permissible to charge loss against other income, including all carrying charges. That is, if the net result is a loss, that loss can be applied to professional income. (This does not apply when purchasing a farm.)*

**Information supplied by the Income Tax Dept.*

(l) Business tax will be allowed as an expense, but Dominion, Provincial or Municipal income tax will not be allowed.*

Convention Expenses*

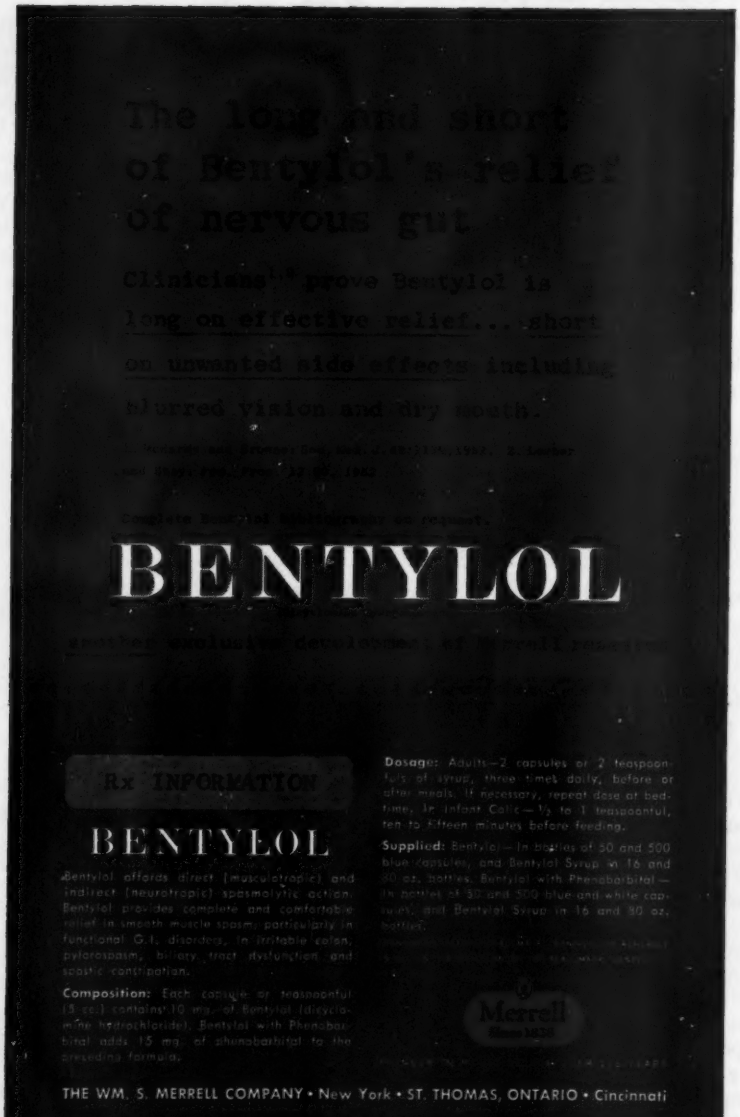
"Effective January 1, 1948, the reasonable expenses incurred by members of the medical profession in attending the following Medical Conventions will be admitted for Income Tax purposes against income from professional fees:

1. One Convention per year of the Canadian Medical Association.

2. One Convention per year of either a Provincial Medical Association or a Provincial Division of the Canadian Medical Association.

3. One Convention per year of a Medical Society or Association of Specialists in Canada or the United States of America.

The expenses to be allowed must be reasonable and must be properly substantiated; e.g., the taxpayer should show (1) dates of the Convention; (2) the number of days present, with proof of claim supported by a certificate of attendance issued by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Rx INFORMATION
BENTYLOL
Bentylol affords direct (musculotropic) and indirect (neurotropic) spasmolytic action. Bentylol provides complete and comfortable relief in smooth muscle spasm, particularly in functional G.I. disorders, in irritable colon, pylorospasm, biliary tract dysfunction and spastic constipation.
Composition: Each capsule or teaspoonful (5 cc.) contains 10 mg. of Bentylol (dicyclomine hydrochloride). Bentylol with Phenobarbital adds 15 mg. of phenobarbital to the preceding formula.
Dosage: Adults—2 capsules or 2 teaspoonfuls of syrup, three times daily, before or after meals. If necessary, repeat dose at bedtime. In Infant Colic—½ to 1 teaspoonful, ten to fifteen minutes before feeding.
Supplied: Bentylol—in bottles of 50 and 500 blue capsules, and Bentylol Syrup in 16 and 30 oz. bottles. Bentylol with Phenobarbital—in bottles of 50 and 500 blue-and-white capsules, and Bentylol Syrup in 16 and 30 oz. bottles.
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the organization sponsoring the meetings; (3) the expenses incurred, segregating between (a) transportation expenses, (b) meals and (c) hotel expenses, for which vouchers should be obtained and kept available for inspection.

None of the above expenses will be allowed against income received by way of salary since such deductions are expressly disallowed by statute."

**As the deduction of these expenses has recently been challenged by the Income Tax Appeal Board, the question of convention expenses is now under review. The memorandum quoted above applies to the taxation year 1954. Its future application depends on the outcome of an appeal which is at present before the Exchequer Court of Canada.*

Professional Men Under Salary Contract

The employees' annual contribution to an approved Pension Plan and alimony payments may be deducted from salary income.

Amendments to the Income Tax Act, introduced in 1951 and made retroactive to the beginning of the calendar year 1951, provide for the deduction of certain expenses from salary income.

The allowable expenses include travelling expenses, annual professional membership dues, office rent, salary to an assistant or substitute and supplies consumed directly in the performance of the duties of employment.

The annual registration fee of the Provincial medical licensing authority would be allowable if paid by the doctor himself.

Certain conditions are attached to the allowance of the expenses and without trying to recite the exact provisions of the law the main points are:

(a) That the expenses must have been incurred in the performance of the duties of the office or employment.

(b) That the employee is required, under the contract of employment, to pay the expenses.

(c) To claim travelling expenses the employee must be ordinarily required to carry on the duties of his employment away from his employer's place of business. Travelling between the doctor's home and his office is not included.

Where the travelling expenses are allowable under these provisions, depreciation may be claimed on the automobile used for this purpose but no other claim for depreciation may be made.

Income From a Partnership

Additional expenses incurred by a partner, but not charged to the partnership, may be claimed as a deduction from the partner's share of income. However, the partner must be in a position to substantiate these expenses, to show why they were not charged directly to the partnership and that they were necessarily laid out to earn the partnership income.

Brandon and District Medical Association

A meeting of the Brandon and District Medical Association was held on Thursday, December 8th. In the forenoon four visitors from the Children's Hospital — Doctors C. C. Ferguson, H. Medovy, J. K. Martin and A. E. Childe visited the Brandon General Hospital and consulted with members of the medical staff on the facilities for paediatric care. Lunch was served at the hospital under the supervision of the Superintendent, Superintendent of Nurses and Dietitian.

A Clinical Session in the afternoon was devoted to presentations by visiting speakers as follows:

Dr. C. C. Ferguson — Surgical Emergencies in the Newborn Period.

Dr. H. Medovy talked on Newborn Nursery Care and gave illustrated demonstration of conditions of paediatric interest.

Dr. A. E. Childe gave an illustrated talk on Diseases of the Bone in Childhood.

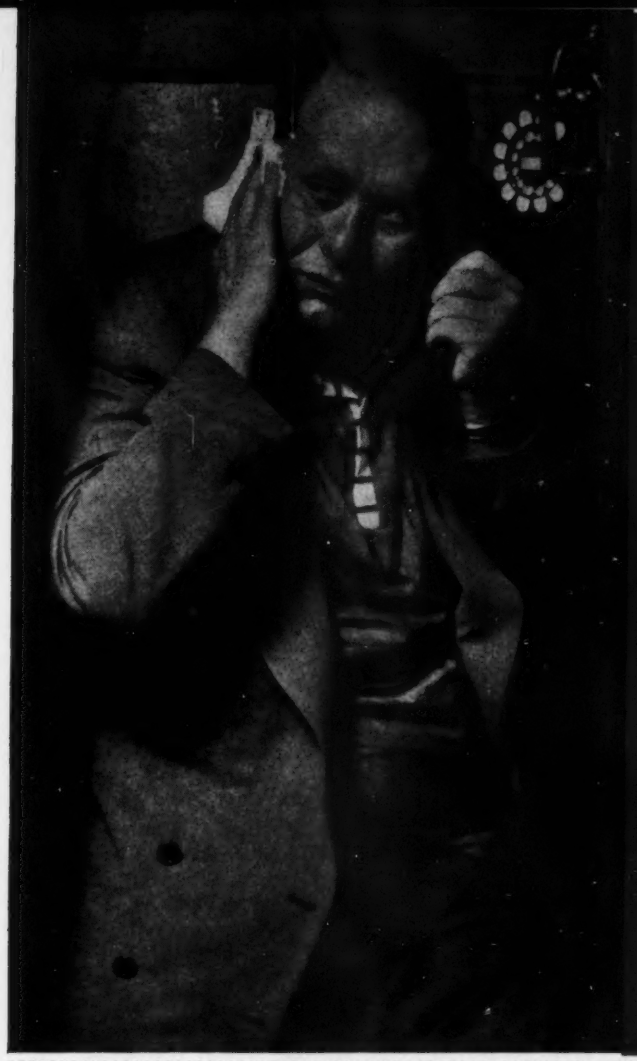
Dr. J. K. Martin talked on Etiology of Cerebral Palsy.

Attending the sessions were: Doctors M. Atkinson, Portage la Prairie; J. B. Baker, Brandon; I. B. Barclay, Brandon; M. E. Bristow, Brandon; R. D. Campbell, Brandon; A. E. Childe, Winnipeg; E. Cziller, Brandon; H. S. Evans, Brandon; C. C. Ferguson, Winnipeg; J. A. Findlay, Winnipeg; F. Fjeldsted, Brandon; D. J. Gill, Brandon; W. K. Hames, Kenton; W. P. Hirsch, Brandon; J. E. Hudson, Hamiota; A. P. Lapko, Brandon; M. T. Macfarland, Winnipeg; C. M. MacLean, Brandon; J. K. Martin, Winnipeg; H. Medovy, Winnipeg; R. F. M. Myers, Brandon; R. O. McDiarmid, Brandon; H. M. McIntyre, Brandon; J. McKenty, Winnipeg; J. D. McMillan, Oak River; R. D. Oatway, Brandon; F. J. E. Purdie, Brandon; J. P. Ramsaran, Brandon; J. E. Rowlands, Brandon; W. Schlichther, Brandon; J. H. Scott, Brandon; W. Shaharhiw, Brandon; V. J. H. Sharpe, Brandon; G. J. Smith, Gladstone; W. E. Yury, Rivers. Following the papers there was a panel discussion in which questions were presented by members of the audience.

A reception and dinner were held in the Prince Edward Hotel, followed by a business meeting addressed by Dr. J. McKenty on behalf of the College of General Practice, Dr. J. A. Findlay, Representative from the Brandon and District Medical Association to the Executive Committee of the Manitoba Medical Association and Dr. M. T. Macfarland, Executive Secretary.

Following the meeting guests were entertained at the home of Doctor and Mrs. R. O. McDiarmid.

M. T. M.



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Ethical Pharmaceuticals of Merit since 1878

Department of Health and Public Welfare

Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1955 Dec. 4 to Dec. 31,'55	1955 Nov. 6 to Dec. 3,'55	1954 Nov. 28 to Dec. 25,'54	1954 Oct. 31 to Nov. 27,'54	Total Jan. 1 to Dec. 31,'55	Total Jan. 1 to Dec. 25,'54
Anterior Poliomyelitis	2	2	0	0	30	117
Chickenpox	125	135	204	252	1262	1966
Diphtheria	4	5	0	0	11	0
Diarrhoea and Enteritis, under 1 year	32	15	5	6	140	164
Diphtheria Carriers	0	1	0	0	3	0
Dysentery—Amoebic	0	0	0	0	0	0
Dysentery—Bacillary	2	1	0	1	31	21
Dysentery—Bacillary Carrier	0	0	0	0	0	1
Erysipelas	1	0	0	0	14	25
Encephalitis	1	0	0	0	1	5
Influenza	12	9	6	2	234	90
Measles	199	196	140	106	2704	1184
Measles—German	4	3	0	3	72	17
Meningococcal Meningitis	5	0	3	2	17	29
Mumps	110	55	111	95	1199	1251
Ophthalmia Neonatorum	0	0	0	0	1	0
Puerperal Fever	0	0	0	0	1	1
Scarlet Fever	13	31	31	42	199	547
Septic Sore Throat	0	10	2	3	31	55
Smallpox	0	0	0	0	0	0
Tetanus	0	0	0	0	0	2
Trachoma	0	1	0	0	1	0
Tuberculosis	56	41	37	60	577	753
Typhoid Fever	0	0	0	0	7	3
Typhoid Paratyphoid	0	0	0	0	0	0
Typhoid Carriers	0	0	0	0	0	0
Undulant Fever	0	1	0	0	8	7
Whooping Cough	18	25	115	105	680	436
Gonorrhoea	104	83	91	124	1214	1361
Syphilis	6	6	5	3	101	93
Jaundice Infectious	33	35	32	26	362	396
Tularemia	0	0	0	0	2	2

Four-week Period December 4th to December 31st, 1955

DISEASES (White Cases Only)	*828,000 Manitoba	*861,000 Saskatchewan	*2,825,000 Ontario	*2,953,000 Minnesota
Anterior Poliomyelitis	2	1	3	14
Chickenpox	125	9	1520	....
Diarrhoea and Enteritis, under 1 Yr.	32	17	....	....
Diphtheria	4	3	....	2
Diphtheria Carriers	....	4	....	....
Dysentery—Amoebic	....	....	....	....
Dysentery—Bacillary	2	3	4	9
Encephalitis, Infectious	1	....	2	....
Erysipelas	1	....	2	....
Influenza	12	....	2	5
Jaundice, Infectious	33	71	52	55
Measles	199	16	2454	16
German Measles	4	....	272	....
Meningitis Meningococcus	5	....	4	2
Mumps	110	....	1279	....
Ophthal. Neonat.	....	....	....	....
Puerperal Fever	....	....	....	....
Scarlet Fever	13	3	307	59
Septic Sore Throat	....	1	2	40
Smallpox	....	....	....	....
Tetanus	....	....	....	....
Trachoma	....	....	....	....
Tuberculosis	56	18	72	121
Tularemia	....	1	....	....
Typhoid Fever	....	....	3	....
Typh. Para. Typhoid	....	1	1	....
Typhoid Carrier	....	....	....	....
Undulant Fever	....	....	....	4
Whooping Cough	18	45	199	26
Gonorrhoea	104	....	122*	....
Syphilis	6	....	16*	....

*Statistics for three weeks only.

DEATHS FROM REPORTABLE DISEASES
December, 1955

Urban—Cancer, 59; Lethargic Encephalitis, 1; Pneumonia, Lobar (490), 6; Pneumonia (other forms), 13; Tuberculosis, 1; Whooping Cough, 1. Other deaths under 1 year, 24. Other deaths over 1 year, 190. Stillbirths, 13. Total, 308.

Rural—Cancer, 30; Influenza, 1; Measles, 1; Pneumonia, Lobar (490), 3; Pneumonia (other forms), 14; Syphilis, 2; Tuberculosis, 2; Whooping Cough, 2; Diarrhoea and Enteritis, 3. Other deaths under 1 year, 17. Other deaths over 1 year, 210. Stillbirths, 12. Total, 297.

Indians—Cancer, 2; Influenza, 1; Pneumonia (other forms), 2; Diarrhoea and Enteritis, 3. Other deaths under 1 year, 4. Other deaths over 1 year, 4. Stillbirths, 2. Total, 18.

♦

This report covers the final four-week period of the year and so gives an opportunity to comment on the full year.

Poliomyelitis figures were down to the usual non epidemic year. No cases under one year and the balance distributed fairly equal up to age 40 — none older. Out of a total of 32 (latest figure) none died, 21 have either no paralysis or made a complete recovery, only three were respirator cases and of these only one remains in a respirator. Not a good year to assess the value of Salk Vaccine!

Diphtheria—"Pride goeth before a fall." We were so proud of our zero figure in 1954 and then eleven cases and three carriers in 1955! None of the cases had been immunized, two of the carriers had and the third was an adult who no doubt had developed an immunity. Shows the value of toxoid! **Immunize!** An upsurge in Diphtheria has occurred in 1955 across Canada and in the U.S.A.

Infectious Jaundice is still a problem. Please report it and when indicated give Gamma Globulin to the non immune contacts.

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